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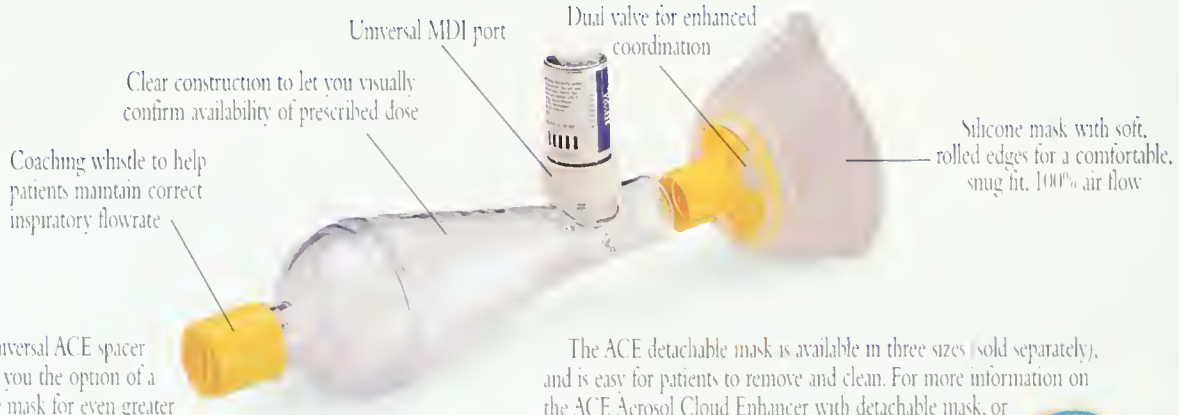
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Contents ...

February 1997
Volume 42, Number 2

IN MEMORIAM

198 Philip Kittredge 1926 - 1996

EDITORIALS

200 Capnography—A Key to Understanding Physiology
by John M Graybeal—Hershey, Pennsylvania

202 Benchmarking Applied to Health Care
by Marvin Lough—Cleveland, Ohio

204 Benchmarking: It's Time To Get on with It
by William H Dubbs—Dallas, Texas

ORIGINAL CONTRIBUTIONS

206 Safety and Efficacy of Oral Levofloxacin versus Cefuroxime Axetil in Acute Bacterial Exacerbation of Chronic Bronchitis
by C Andrew DeAbate, Melvin Russell, Phillip McElvaine, Henry Faris, John Upchurch, Cynthia L Fowler, Elaine M Polak, and Nancy S Morgan—A Multicenter Trial, New Orleans, Louisiana

CASE REPORTS

214 Acute Respiratory Failure from Neck Hematoma Following Venous Cannulation in a Patient Receiving Streptokinase
by Andreja Sinkovič—Maribor, Slovenia

218 CO_2 Retention Secondary to Laparoscopic Cholecystectomy
by David F Mayo, Edwin C Kohl, and Charles G Durbin Jr—Charlottesville, Virginia

COMMENTARIES

221 Benchmark Data for the Outcome of Mechanical Ventilation in a Pediatric Intensive Care Unit
by Robert L Chatburn, Mohamad El-Khatib, and Jeffrey L Blumer—Cleveland, Ohio

GRAPHICS CORNER

227 Abnormal Capnogram during Laparoscopic Cholecystectomy in a Lung-Transplant Patient
by Janice Benedetti and Charles G Durbin Jr—Charlottesville, Virginia

PFT CORNER

230 Bronchodilator Response in the 'Borderlands'
by Mark Nicolls, Jack Wanger, Charles G Irvin—Denver, Colorado

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. . . Contents

February 1997
Volume 42, Number 2

Correction

226 Corrected Author Name
in Manual Volume-Controlled Ventilation in Preterm Neonates Compared to Ventilation via Self-Inflating Resuscitator [Respir Care 1996;41(12):1083-1089].

Books, Films, Tapes, & Software

233 Statistics for the Social Sciences
Reviewed by Robert C Shaw—Lenexa, Kansas

234 Pulmonary and Critical Care Pharmacology and Therapeutics
Reviewed by Hugh S Mathewson—Kansas City, Kansas

Letters

236 A Problem with Bear Cubs and Isothermal Circuits?
by Michael S Lasky—North Staten Island, New York
response by Damon R Lawson, Rick Uhlemann, and Brian Pierro—Riverside, California

237 Observations on the Therapist's Role
by Greg Hagan—Madisonville, Kentucky

Historical Note

238 Effective Inhalation Therapy: Bronchial Asthma
by Edwin R Levine—Reprinted from Effective Inhalation Therapy. Chicago: National Cylinder Gas Co, 1953. Submitted by Teri Nikolai Wilson—Centerville, Ohio

In This Issue

243 AARC Membership Application
184 Abstracts from Other Journals
256 Advertisers Index & Help Lines
256 Author Index
247 Calendar of Events
241 Call for Open Forum Abstracts
252 Manuscript Preparation Guide
245 MedWatch
250 New Products & Services
248 Notices

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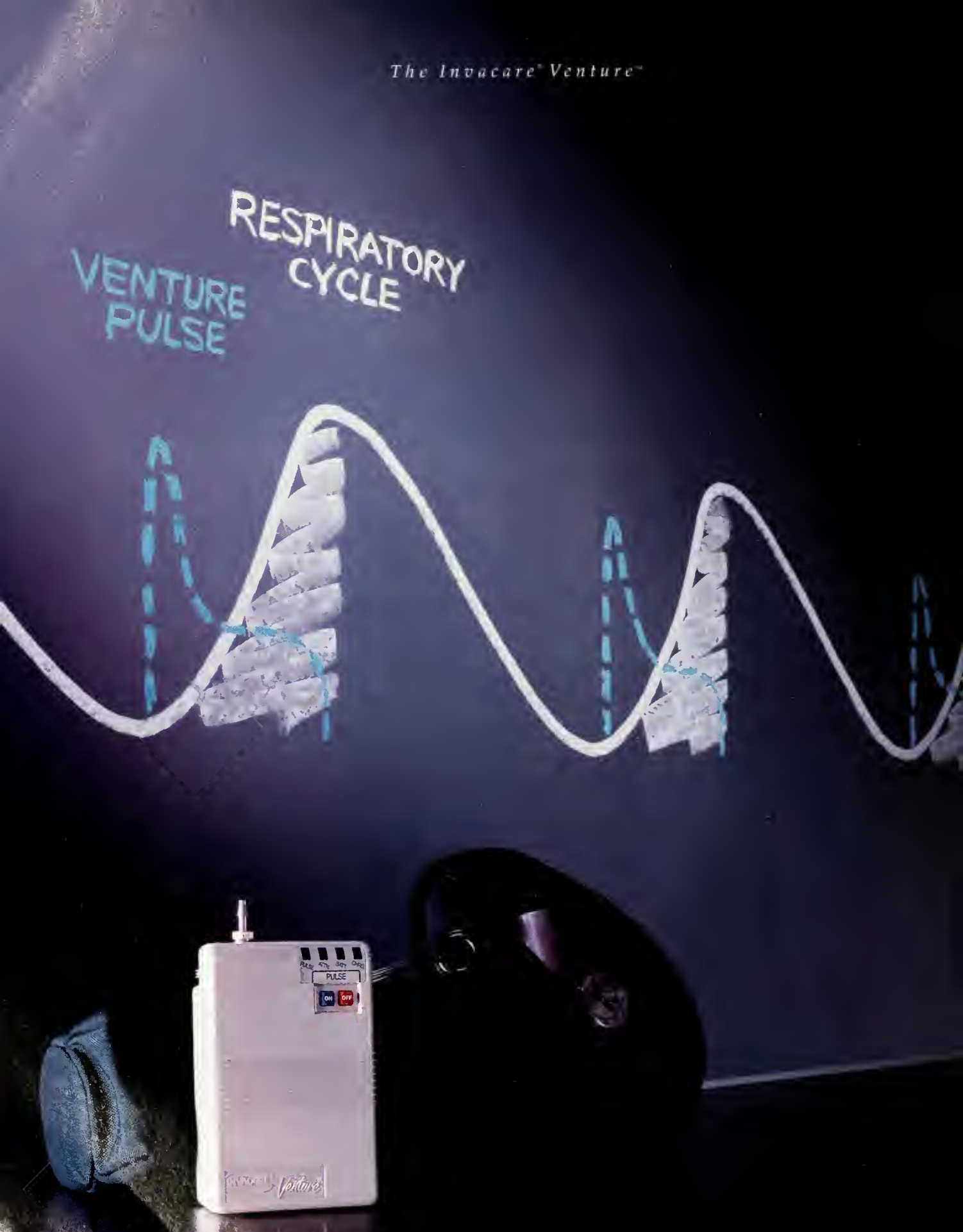

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Editorials, Commentaries, and Reviews To Note

Cystic Fibrosis (State of the Art)—PB Davis, M Drumm, MW Konstan. Am J Respir Crit Care Med 1996;154:1229-1256.

Safety of Vaccinations: Miss America, the Media, and Public Health (Article)—GL Freed, SL Katz, SJ Clark. JAMA 1996;276(23):1869.

Vaccines and Risks: The Responsibility of the Media, Scientists, and Clinicians (Editorial)—V Cohn. JAMA 1996;276(23):1917-1918.

A Comparison of the Acute Physiology and Chronic Health Evaluation (APACHE) II Score and the Trauma-Injury Severity Score (TRISS) for Outcome Assessment in Intensive Care Unit Trauma Patients—DT Wong, PM Barrow, M Gomez, GP McGuire. Crit Care Med 1996;24 (10):1642.

OBJECTIVE: To assess the ability of the Acute Physiology and Chronic Health Evaluation (APACHE II) system and Trauma-Injury Severity Scoring (TRISS) system in predicting group mortality in intensive care unit (ICU) trauma patients. DESIGN: Prospective study. SETTING: A Canadian adult trauma tertiary referral hospital. PATIENTS: Consecutive trauma patients admitted to the medical-surgical ICU or the neurosurgical ICU. INTERVENTION: None. MEASUREMENTS & MAIN RESULTS: For each patient, demographic data, mechanism of injury, and surgical status were collected. Revised Trauma Scores and Injury Severity Scores were calculated from emergency room and operative data. The APACHE II score was calculated based on the data from the first 24 hours of ICU admission. The probability of death was calculated for each patient based on the APACHE II and TRISS equations. The ability to predict group mortality for APACHE II and TRISS was assessed by receiver operating characteristic curve analysis, two-by-two decision matrices, and calibration curve analysis. Four hundred seventy trauma patients were admitted to the ICU. Sixty-three (13%) patients died and 407 (87%) survived. There were significant differences between survivors and nonsurvivors in age, Glasgow Coma Scale, Revised Trauma Score, Injury Severity Score, and APACHE II score. By receiver operating characteristic curve analysis, the areas under the curves (± SEM) of APACHE II and TRISS were 0.92 ± 0.02 and 0.89 ± 0.02, respectively. Using two-by-two decision matrices with a decision criterion of 0.5, the sensitivities, specificities, and percentages correctly classified were 50.8%, 97.3%, and 91.1%, respectively, for APACHE II, and 50.8%, 97.1%, and 90.9%, respectively, for TRISS. From the calibration curves, the r^2 value was 0.93 ($p = 0.0001$) for APACHE II and 0.67 ($p = 0.004$) for TRISS. CONCLUSIONS: Both APACHE II and TRISS scores were shown to accurately predict group mortality in ICU trauma patients. APACHE II and TRISS may be utilized for quality assurance in ICU trauma patients. However, neither APACHE II nor TRISS provides sufficient confidence for prediction of outcome of individual patients.

Lung Volume Reduction Surgery in Ventilator-Dependent COPD Patients—GJ Criner, G O'Brien, S Furukawa, F Cordova, M Swartz, M Fallahnejad, G D'Alonzo. Chest 1996;110(4):877.

BACKGROUND: Lung volume reduction surgery has been advocated recently as adjunctive surgical therapy to improve lung and chest wall mechanics in selected patients with diffuse emphysema. Although clear-cut guidelines to select candidates have not been fully established, patients decompensated with significant pulmonary artery hypertension and hypercapnic respiratory failure are currently not considered suitable subjects. Accordingly, ventilator-dependent COPD patients are not considered candidates for this procedure. However, because ventilator-dependent COPD patients have an exceptionally poor prognosis, we elected to offer them this promising, but unproved surgical intervention. Herein, we describe the outcome of these 3 patients. PATIENTS: The 3 patients had recurrent exacerbations of COPD precipitating respiratory failure, and following aggressive medical therapy remained mechanically ventilated for 11 to 16 weeks (1 patient had a brief period of successful weaning before returning to mechanical ventilation). Prior to surgery, the patients had severe hypercapnia and cor pulmonale. Compared with preoperative values, surgery resulted in improvements in P_{aO_2}/F_{IO_2}, 304 ± 80 (SD) versus 229 ± 48 mm Hg, reductions in P_{aCO_2}, 44 ± 3 versus 60 ± 9 mm Hg, increases in FVC, 1.63 ± 0.52 versus 1.09 ± 0.05 L, and maximum inspiratory pressure, 57 ± 22 versus 29 ± 12 cm H_2O. Postoperative complications included persistent air leaks and 1 tension pneumothorax. Patients weaned from mechanical ventilation after 10 to 21 days all were discharged home and they continue to demonstrate improved gas exchange and functional status. CONCLUSIONS: Lung volume reduction surgery in select, ventilator-dependent COPD patients can result in improved gas exchange and respiratory mechanics that enable successful weaning and overall improved functional status.

Economic Aspects of Lung Volume Reduction Surgery—RK Albert, S Lewis, D Wood, JO Benditt. Chest 1996;110(4):1068.

OBJECTIVE: To investigate the economics of lung volume reduction surgery. DESIGN: Medical center and physician charges obtained from billing records. SETTING: Academic health center. PATIENTS: Twenty-three consecutive patients undergoing lung volume reduction surgery at a single institution who were discharged from the hospital prior to November 1, 1995. OUTCOME MEASURES: Length of hospital stay, mortality, medical center charges and professional fees, and sponsor reimbursement. RESULTS: Median hospital stay was 8.0 days and there were no deaths. The median charge was $26,669 (range, $20,032 to $75,561) of which 73% was for medical center services and 27% was for physician services. Fees for medical center rooms and operating suite time accounted for 71% of medical center charges. Charges by surgeons and anesthesiologists accounted for 77% of professional fees. Total charges were directly related to length of

stay ($r^2 = 0.95$). Median reimbursement for medical center services was $22,264 (114%; range, $13,333 to $123,362) and for physician services was $2,783 (34%; range, $2,597 to $11,265), resulting in a median total reimbursement that represented 94% of total charges. The median reimbursement-to-cost ratio was 1.22, compared with 1.05 for all medical services in fiscal year 1995. CONCLUSIONS: These data must now be assessed relative to outcomes such as quality of life, patient function, and long-term survival to determine cost-effectiveness of lung volume reduction surgery.

Acute Response to Inhaled Nitric Oxide in Newborns with Respiratory Failure and Pulmonary Hypertension—RW Day, JM Lynch, KS White, RM Ward. Pediatrics 1996;98(4):698.

OBJECTIVE: Systemic oxygenation is improved by inhaled nitric oxide therapy in some newborns with respiratory failure and pulmonary hypertension. Our results with inhaled nitric oxide were reviewed to determine factors associated with an acute improvement in systemic oxygenation. METHODS: Newborns with oxygenation indices of 25 to 40 were prospectively randomized to receive conventional therapy with or without 20 ppm inhaled nitric oxide. All newborns with oxygenation indices > 40 were treated with inhaled nitric oxide. Hemodynamic, blood gas, and Doppler ultrasound measurements were performed before and after 30 to 60 minutes of observation or therapy. The severity of lung disease was classified by the chest radiograph as: (1) normal or focal disease; (2) moderate diffuse disease—diffuse lung disease with well-defined heart borders; or (3) severe diffuse disease—diffuse lung opacification with indistinct heart borders. RESULTS: Heart rate, blood pressure, and ductal diameters did not change. Blood gases and ductal shunting acutely improved only in patients treated with inhaled nitric oxide. Patients with normal lung fields or focal disease had the greatest degree of improvement in systemic oxygenation. Changes in oxygenation were not influenced by gestational age, baseline blood gases, the proportion of right-to-left ductal shunting, prior treatment with a surfactant, or the use of conventional or high-frequency jet ventilation. Collectively, blood gases and ductal shunting did not improve with inhaled nitric oxide in patients with lung hypoplasia or severe diffuse lung disease. Sustained improvement in oxygenation occurred in 87% of patients with oxygenation indices > 40 in whom oxygenation indices < 40 acutely developed after exposure to nitric oxide, whereas 90% of patients in whom oxygenation indices < 40 did not acutely develop were treated with extracorporeal membrane oxygenation or ultimately died. CONCLUSIONS: Inhaled nitric oxide acutely improves systemic oxygenation in many newborns with respiratory failure and pulmonary hypertension. The diagnosis and chest radiograph are helpful in identifying patients who will have favorable acute responses to therapy. In patients with severe hypoxemia, the need for invasive support with extracorporeal membrane oxygenation may be determined by an acute trial of inhaled nitric oxide.

Four Patterns of Response to Inhaled Nitric Oxide for Persistent Pulmonary Hypertension of the Newborn—AP Goldman, RC Tasker, SG Haworth, PE Sigston, DJ Macrae. Pediatrics 1996;98(4):706.

OBJECTIVE: To determine the clinical role of inhaled nitric oxide (iNO) in the treatment of persistent pulmonary hypertension of the newborn (PPHN). STUDY DESIGN: Prospective open observational clinical study. SETTING: A regional cardiac and pediatric intensive care unit. METHODS: Twenty-five consecutive near-term neonates (> 35 weeks gestation) with severe PPHN (oxygenation index [OI] > 25) were given a trial of iNO of 20 ppm for 20 minutes. Neonates who showed a > 20% improvement in P_{aO_2} as well as a decrease in the OI to below 40 were defined as responders and continued on this therapy. RESULTS: Four patterns of response emerged to the iNO therapy: Pattern 1 neonates (n = 2) did not respond to the initial trial of iNO—1 survived. Pattern 2 neonates (n = 9) responded to the initial trial of iNO, but failed to sustain this response over 36 hours, as defined by a rise in the OI to > 40. Six survived, 5 with extracorporeal membrane oxygenation. Pattern 3 neonates (n = 11) responded to the initial trial of iNO, sustained this response, and were successfully weaned from iNO within 5 days—all survived to discharge. Pattern 4 neonates (n = 3) responded to the initial trial of iNO, but developed a sustained dependence on iNO for 3 to 6 weeks. All 3 died and lung histology revealed severe pulmonary hypoplasia and dysplasia. These neonates (pattern 4) not only required iNO for a longer period of time than did the sustained responders (pattern 3), but they required significantly higher doses of iNO during their first 5 days of iNO therapy. CONCLUSIONS: Early responses to iNO may not be sustained. Neonates with pulmonary hypoplasia and dysplasia may have a decreased sensitivity and differing time course of response to iNO when compared with patients who have PPHN in fully developed lungs.

Planning Patient Services for Intermediate Care Units: Insights Based on Care for Intensive Care Unit Low-Risk Monitor Admissions—JE Zimmerman, DP Wagner, X Sun, WA Knaus, EA Draper. Crit Care Med 1996;24(10):1626.

OBJECTIVE: To describe the technology and nursing services that would be required to care for intensive care unit (ICU) low-risk monitor admissions in an intermediate unit. DESIGN: Prospective, multicenter, inception cohort analysis. SETTING: Forty U.S. hospitals with > 200 beds, including 26 hospitals that were randomly selected and 14 that volunteered for the study. PATIENTS: A sample of 8,040 ICU patients admitted to the ICU for monitoring, who received no active life-support treatment on ICU Day 1. INTERVENTIONS: None. MEASUREMENTS & MAIN RESULTS: Demographic, physiologic, and treatment information were obtained during ICU days 1 to 7. A previously validated multivariate equation was used to identify 6,180 monitor admissions at low (< 10%) risk for receiving active treatment during their entire ICU stay. We used daily Therapeutic Intervention Scoring System (TISS) data to identify the equipment, type and amount of nursing care, and the types of active treatment that would have been used had these ICU patients been admitted to an intermediate care unit. Mean Day-1 ICU TISS scores were as follows: 16.4 for all patients; 18.3 for surgical patients; and 13.5 for medical admissions. Concentrated nursing care accounted for 89% and technologic monitoring for 11% of Day-1 TISS points. Surgical admissions had a 2.8-day mean ICU length of stay and received an average of 16.5 TISS points/patient/day. Medical admissions had a 2.7-day mean ICU length of stay and received an average of 12.3 TISS points/patient/day. Subsequent active life-support therapy was received by 4.4% of these ICU low-risk monitor admissions. CONCLUSIONS: The services received by ICU low-risk monitor admissions provide insight regarding the equipment and nursing care that might be required, and the kinds of emergencies that might occur, if these patients were cared for in medical and surgical intermediate care units. Our data suggest that if ICU low-risk monitor patients were admitted to an intermediate care unit, they would mainly require concentrated nursing care (nurse/patient ratio of 1:3 to 1:4) and limited technologic monitoring.

Decline in Hospital Utilization and Cost Inflation under Managed Care in California—JC Robinson. JAMA 1996;276(13):1060.

OBJECTIVE: To measure the impact of health maintenance organizations (HMOs) on hospital capacity, utilization, and expenditures between 1983 and 1993. DESIGN: Multivariate regression analysis. SETTING: Private nonprofit and for-profit hospitals in California with 25 or more beds. PATIENTS: Patient discharge abstract data were used to measure growth of HMO penetration of local hospital markets. INTERVENTIONS: None. MAIN OUTCOME MEASURES: Hospital closures, changes in bed capacity, changes in acute care admissions and length of stay, psychiatric inpatient days, subacute inpatient days, inpatient and outpatient surgical procedures, ambulatory patient visits, and hospital expenditures. RESULTS: Between 1983 and 1993 hospital expenditures grew 44% less rapidly in markets with high HMO penetration than in markets with low HMO penetration. Of this, 28% was due to reductions in the volume and mix of services, 6% was due

to reductions in bed capacity, and 10% was due to changes in the intensity of services provided. Health maintenance organizations accelerated the substitution of outpatient for inpatient surgery, the shift from acute to subacute inpatient days, and the reduction of psychiatric hospitalization. CONCLUSION: Managed care is shifting the acute care hospital from the center toward the periphery of the health care system.

The Effects of Patient Volume and Level of Care at the Hospital of Birth on Neonatal Mortality—CS Phibbs, JM Bronstein, E Buxton, RH Phibbs. JAMA 1996;276(13):1054.

OBJECTIVE: To examine the effects of neonatal intensive care unit (NICU) patient volume and the level of NICU care available at the hospital of birth on neonatal mortality. DESIGN: Birth certificate data linked to infant death certificates and to infant discharge abstracts were used in a logistic regression model to control for differences in each patient's clinical and demographic risks. Hospitals were classified by the level of NICU care available (no NICU; level I; intermediate NICU; level II; expanded intermediate NICU; level II+; tertiary NICU; level III) and by the average patient census in the NICU. SETTING: All nonfederal hospitals in California with maternity services. PATIENTS: All births in nonfederal hospitals in California in 1990 (n = 594,104), 473,209 (singletons only) of which were successfully linked with discharge abstracts. Of these infants, 53,229 were classified as likely NICU admissions. MAIN OUTCOME MEASURES: Death within the first 28 days of life, or within the first year of life, if continuously hospitalized. RESULTS: Patient volume and level of NICU care at the hospital of birth both had significant effects on mortality. Compared with hospitals without an NICU, infants born in a hospital with a level III NICU with an average NICU census of at least 15 patients/day had significantly lower risk-adjusted neonatal mortality (odds ratio, 0.62; 95% confidence interval, 0.47-0.82; p = 0.002). Risk-adjusted neonatal mortality for infants born in smaller level III NICUs, and in level II+ and level II NICUs, regardless of size, was not significantly different from hospitals without an NICU, and was significantly higher than hospitals with large level III NICUs. CONCLUSIONS: Risk-adjusted neonatal mortality was significantly lower for births that occurred in hospitals with large (average census, > 15 patients/day) level III NICUs. Despite the differences in outcomes, costs for the birth of infants born at hospitals with large level III NICUs were not more than those for infants born at other hospitals with NICUs. Concentration of high-risk deliveries in urban areas in a smaller number of hospitals that could provide level III NICU care has the potential to decrease neonatal mortality without increasing costs.

A New Microtransducer Catheter for Measuring Esophageal Pressure in Infants—M Gappa, E Jackson, L Pilgrim, K Costeloe, J Stocks. Pediatr Pulmonol 1996;22(2):117.

Measurement of esophageal pressure, as a reflection of pleural pressure, is essential for assessment of dynamic lung mechanics in neonates and infants. Conventionally, an esophageal balloon or a fluid-filled catheter is used, but considerable skill is required to obtain accurate results. Both devices have problems, and failure to achieve valid occlusion tests have been reported, particularly in small infants with lung disease. Recently, a flexible #3 French gauge (FG) microtransducer cathether (MTC®, Dräger, Netherlands) has become available for medical monitoring. We have assessed the accuracy and feasibility of using this device for measuring lung mechanics in 51 spontaneously breathing infants and small children aged 1 day to 24 months (weight 1.35 to 12.0 kg), 9

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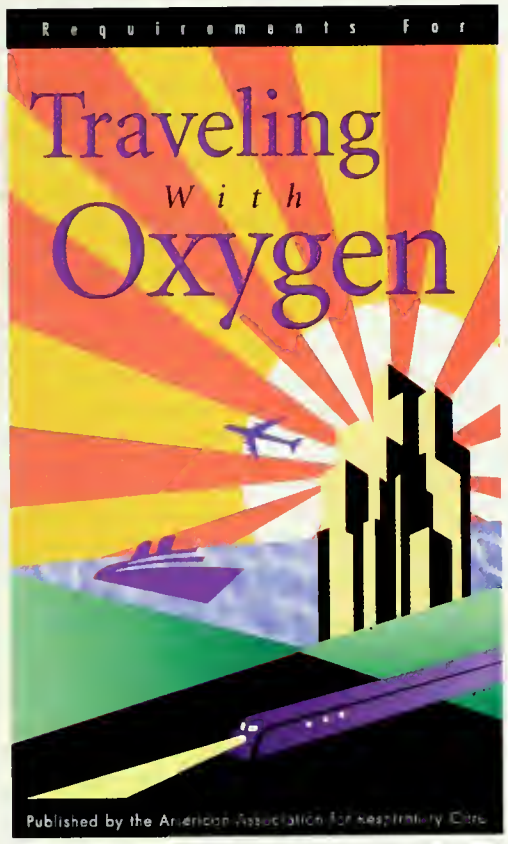

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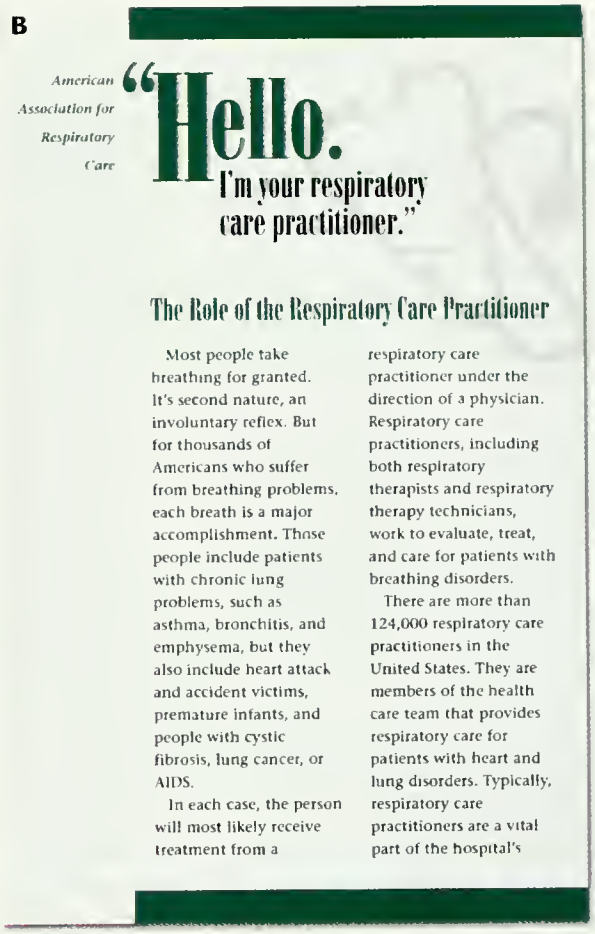
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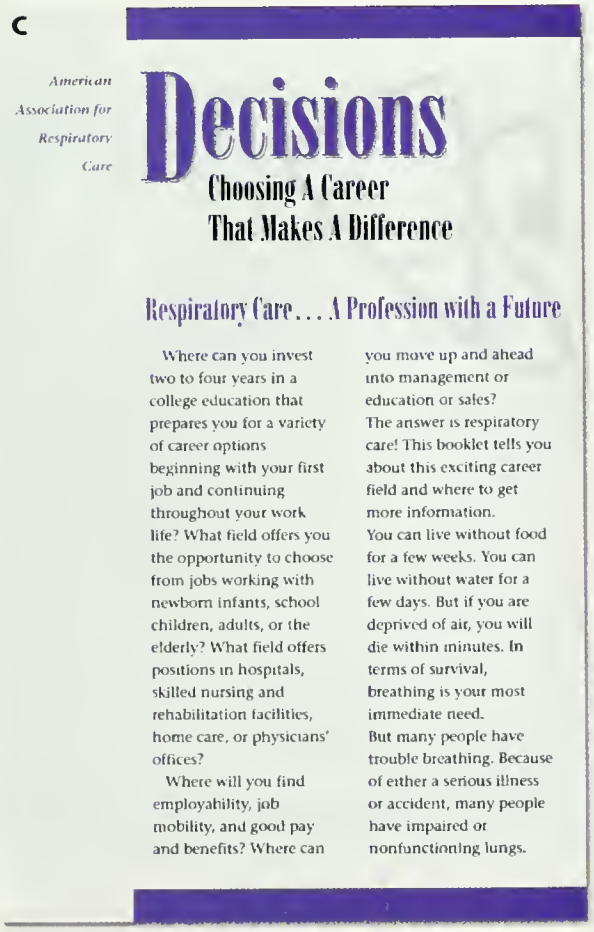
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of whom were healthy neonates, the remainder suffering from a variety of cardiorespiratory diseases, and in 18 sick ventilated infants (weight 0.6 to 4.0 kg). Positioning of the catheter was well tolerated by all infants. The ratio of esophageal to airway opening pressure changes (ΔP_{es}:ΔP_{ao}) ranged from 0.94 to 1.09 [mean (SD) 1.013 (0.03)] for the spontaneously breathing infants and from 0.98 to 1.06 [mean (SD) 1.003 (0.02)] in the ventilated infants with no significant difference in this ratio between the 2 groups ($p = 0.16$). This new generation of catheter tip pressure transducers may provide a simpler and more reliable tool for assessing transpulmonary pressure changes in infants than has previously been available.

Long-Term Effects of Nasal Intermittent Positive-Pressure Ventilation on Pulmonary Function and Sleep Architecture in Patients with Neuromuscular Diseases—F Barbé, MA Quera-Salva, J de Lattre, P Gajdos, AGN Agustí. Chest 1996;110(5):1179.

OBJECTIVE: This article evaluates the long-term clinical and physiologic effects of nocturnal nasal intermittent positive-pressure ventilation (NIPPV) in patients with neuromuscular disease. METHODS: Before and after 18 ± 2 months of NIPPV, we measured during the daytime arterial blood gases, lung mechanics, and respiratory muscle strength in 8 patients (51 ± 5 years; mean ± SEM). Sleep parameters were also evaluated at 10 ± 2 months. RESULTS: All patients tolerated NIPPV, and none required hospitalizatioo during follow-up. After NIPPV, daytime arterial P_{O_2} increased (71 ± 4 to 81 ± 2 mm Hg; $p < 0.05$) and arterial P_{CO_2} decreased (46 ± 3 to 41 ± 1 mm Hg; $p < 0.05$). The change of P_{aO_2} after NIPPV was related to its baseline value ($r^2 = 0.78$, $p < 0.05$). Vital capacity (50 ± 6% predicted), total lung capacity (63 ± 4% predicted), alveolar-arterial oxygen gradient (20 ± 3 mm Hg), and maximal inspiratory (39 ± 9% predicted) or expiratory (32 ± 5% predicted) pressures did not change after NIPPV. The apnea-hypopnea index fell from 22 ± 6 to 1 ± 1 ($p < 0.05$), and both sleep architecture and sleep efficiency (from 59 ± 8% to 83 ± 5% $p < 0.05$) were enhanced. The time spent with an arterial oxygen saturation (S_{aO_2}) value below 90% decreased from 160 ± 53 minutes to 8 ± 4 minutes ($p < 0.05$). Mean (88 ± 3 to 95 ± 1%; $p < 0.05$) and minimal nocturnal S_{aO_2} (67 ± 5 to 89 ± 1%; $p < 0.001$) improved after NIPPV. CONCLUSIONS: In patients with neuromuscular disease, long-term NIPPV is well tolerated and easy to implement clinically. In these patieots, long-term NIPPV improves daytime arterial blood gas values and sleep-disordered breathing. However, it does not modify lung mechanics or respiratory muscle strength.

Perioperative Respiratory Complications in Patients with Asthma—DO Warner, MA Warner, RD Barnes, KP Offord, DR Schroeder, DT Gray, JW Yunginger. Anesthesiology 1996; 85(3):460.

BACKGROUND: Patients with asthma are thought to be at high risk for pulmonary complications to develop during the perioperative period, and these complications may lead to serious morbidity. Existing medical records were reviewed to determine the frequency of and risk factors for perioperative pulmonary complications in a cohort of residents of Rochester, Minnesota, who had asthma and who underwent anesthesia and surgery at the Mayo Clinic in Rochester. METHODS: Medical records were reviewed for all residents of Rochester, Minnesota, who were initially diagnosed as having definite asthma according to strict criteria from 1 January 1964 through 31 December 1983, who subsequently had at least 1 surgical procedure involving a general anesthetic or central neuroaxis block at the Mayo Clinic (n = 706). RESULTS: Bronchospasm was documented in the perioperative records of 12 patients (1.7% [exact 95% confidence interval, 0.9 to 3%]). Postoperative respiratory failure developed in 1 of these patients. Laryngospasm developed in 2 additional patients during operation. All episodes of bronchospasm and laryngospasm in the immediate perioperative period were treated successfully. No episodes of pneumothorax, pneumonia, or death in the hospital were noted. For univariate analysis, characteristics associated with complications included the recent use of antiasthmatic drugs, recent asthma symptoms, and recent therapy in a medical facility for asthma. Patients in whom complications developed were significantly older at diagnosis and at surgery. CONCLUSIONS: The frequency of perioperative bronchospasm and laryngospasm was surprisingly low in this cohort of persons with asthma. These complications did not lead to severe respiratory outcomes in most patients. The frequency of complications was increased in older patients and in those with active asthma. *See the related editorial:* ***Anesthesia for Patients with Asthma: Low Risk but Not No Risk****. MJ Bishop, FW Cheney. Anesthesiology 1996;85(3):455-456.*

Effects of 12-Week Administration of Dornase Alfa in Patients with Advanced Cystic Fibrosis Lung Disease—K McCoy, S Hamilton, C Johnson, for the Pulmozyme Study Group. Chest 1996;110(4):889.

OBJECTIVE: The 12-week efficacy and safety of aerosolized recombinant human DNase (dornase alfa) were evaluated in previously untreated patients with cystic fibrosis (CF) with advanced lung disease. DESIGN: In this multicenter, double-blind, placebo-controlled study, CF patients with advanced lung disease were randomized to receive either dornase alfa or placebo once a day for 12 weeks. PATIENTS: A total of 320 patients in clinically stable condition with documented CF and an FVC < 40% of predicted were recruited from 65 CF Foundation care centers in the United States. The dornase alfa and placebo groups were comparable with respect to age (range, 7 to 57 years), height, and weight. Male subjects outnumbered female subjects (55% vs 45%) and few subjects were younger than 17 years of age (15%). The percentages of predicted FEV_1 and FVC were significantly lower in the dornase alfa group at baseline ($p \leq 0.05$). INTERVENTIONS: Patients were randomly assigned to receive either 2.5 mg dornase alfa once daily (n = 158) or placebo once daily (n = 162). All patients continued to receive standard medications and treatments administered for CF. MEASUREMENTS & RESULTS: Dornase alfa improved the mean percent change in FEV_1 from baseline by 9.4% compared with 2.1% for placebo ($p < 0.001$). The actively treated group showed a 12.4% improvement in FVC compared with 7.3% for placebo ($p < 0.01$). There were no differences between the treatment groups in dyspnea score, number of days receiving I.V. antibiotics, or length of hospital stay; the overall incidence of adverse events was comparable between treatment groups. Fifteen patients died: 9 in the dornase alfa group and 6 in the placebo group; no differentiating clinical characteristics were demonstrated. CONCLUSIONS: Pulmonary function as measured by FEV_1 and FVC improved significantly in the dornase alfa-treated patients. Dornase alfa was found to be safe and well tolerated over the 12-week study period. *See the related editorial:* ***Dornase Alfa Therapy in Cystic Fibrosis: Who Should Get It?*** *JF Kanga. Chest 1996; 110(4):871-872.*

Predicting Eventual Success or Failure to Wean in Patients Receiving Long-Term Mechanical Ventilation—EH Gluck, L Corgian. Chest 1996; 110(4):1018.

PURPOSE: To determine if the ultimate ability of a long-term ventilated patient to wean can be predicted at the time of his or her admission to a

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long-term ventilator unit. DESIGN: Two-phased prospective study. SETTING: Long-term ventilator facility, university-affiliated. SUBJECTS: Adults ventilated for an average of 3 weeks, who did not have sepsis and who did not have chest tubes or progressive neurologic impairment. INTERVENTIONS: On admission to the long-term ventilator unit, historic factors, ventilator history, and the following laboratory and metabolic tests were obtained: electrolytes, serum calcium, magnesium, and phosphorus, WBC, hemoglobin, albumin, total protein, transferrin, oxygen consumption, carbon dioxide production, respiratory quotient, and dead space/tidal volume. The patients were then placed in a weaning protocol utilizing increasing duration of pressure support ventilation during the day with complete rest at night. Forty-two days after enrollment in the study, representing three times the duration of the weaning protocol, the patients who successfully weaned were compared to those who remained ventilator dependent (n = 20). Patients who died or were transferred to another institution were excluded from this phase of the study, because we were trying to develop parameters that would be predictive of successful weaning. A parameter was considered to be predictive, and retained for the scoring system, if it produced at most 15% false-positives and false-negatives. A score of 0 was then assigned to the threshold value that produced no false-positives; 2 to the threshold value that produced no false-negatives and 1 to the intermediate values. The scoring system was then applied to a new prospective group of patients (n = 72). MEASUREMENTS & MAIN RESULTS: Of all the parameters evaluated, only the following satisfied the false-positive and false-negative requirements; static compliance, airway resistance, dead space to tidal volume ratio, P_{aCO_2}, and frequency/tidal volume. Applying these, in the scoring system, to the initial group of patients, demonstrated that a score > 3 was associated with failure to wean; a score < 3 was associated with successful weaning, and a score of 3 was not predictive. Using these thresholds, the data were applied to the new prospective group of patients, which again demonstrated that a score of > 3 was associated with failure to wean in all cases. A score < 3 was again associated with successful weaning but there were 2 false-positives. The sensitivity, specificity, and positive predictive and negative predictive values for the scoring system were 1.0, 0.91, 0.83, and 1.0, respectively. None of the individual parameters included in the scoring system demonstrated equivalent statistical results. All but 2 of the patients who died prior to finishing the weaning period had weaning scores, which suggested that they would not be successfully weaned. CONCLUSIONS: Parameters that are generally available, when combined into a scoring system, can predict at the time of admission to a long-term ventilator unit, in most cases, whether a patient will eventually wean. The scoring system resulted in no false-negatives and an acceptable number of false-positives. None of the individual parameters were as reliable as the scoring system as a whole.

Association between Reduced Cuff Leak Volume and Postextubation Stridor—RL Miller, RP Cole. Chest 1996;110(4):1035.

OBJECTIVE: Laryngotracheal injury or edema in the setting of intubation may narrow the upper airway and predispose toward postextubation stridor. The presence or absence of an audible air-leak when the sealing balloon cuff of the endotracheal tube is deflated has been demonstrated to be a marker of laryngotracheal edema in high-risk patients. We hypothesized that (1) The volume of the cuff leak can be quantified in a general medical ICU population, and (2) the cuff leak volume can be correlated with likelihood of postextubation stridor. METHODS: Within 24 hours of both the initiation and termination of mechanical ventilation, the cuff leak volume, defined as the difference between the inspiratory tidal volume and the averaged expiratory tidal volume while the cuff around the endotracheal tube was deflated, was recorded. RESULTS: In 100 consecutive intubations, the pre-extubation cuff leak

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volume was 349 ± 163 mL [mean ± SD]). Overall, 6% of extubations were accompanied by postextubation stridor. The mean cuff leak volume measured within 24 hours of planned extubation was significantly lower in those who subsequently developed stridor in comparison to those who did not (180 ± 157 mL vs 360 ± 157 mL; p = 0.012). The positive predictive value for postextubation stridor in the setting of a cuff leak < 110 mL was 0.80, the predictive value for absence of postextubation stridor with a cuff leak volume > 110 mL was 0.98, and the specificity of the test was 0.99. No other demographic factors or indexes related to mechanical ventilation were significantly different between the 2 groups. CONCLUSION: A reduced cuff leak volume prior to extubation identifies a population at increased risk for postextubation stridor.

Eliminating Sleep-Associated Hypoxemia Improves Growth in Infants with Bronchopulmonary Dysplasia—LJ Moyer-Mileur, DW Nielson, KD Pfeffer, MK Witte, DL Chapman. Pediatrics 1996;98(4):779.

OBJECTIVE: Infants with bronchopulmonary dysplasia (BPD) have been previously reported to have a decrease in growth velocity after stopping supplemental oxygen (SO). SO was stopped after a short-term recording (20-30 min) of pulse oxygen saturation (S_{aO_2}) of 92% or greater in room air. Other studies have documented that S_{aO_2} decreases further during feedings and sleep in infants with BPD. Two questions were asked: (1) whether short-term, awake S_{aO_2} studies would reliably predict prolonged sleep S_{aO_2}; and (2) how S_{aO_2} sustained at 88% to 91% versus 92% or greater in room air would impact growth velocity in infants with BPD. METHODOLOGY: Short-term S_{aO_2} studies were prospectively compared with prolonged sleep S_{aO_2} (n = 63) and the growth velocity of infants who had SO discontinued after a prolonged sleep S_{aO_2} recording of 88% to 91% (Group 1; n = 14) versus 92% or greater (Group 2; n = 34) in room air. RESULTS: Failure to maintain S_{aO_2} at predetermined levels occurred in 18 (29%) of 63 infants during their first prolonged sleep study. There was no correlation between short-term awake S_{aO_2} and prolonged sleep S_{aO_2} recordings (r = 0.02). Body weight, height, weight for height, and rate of weight gain were similar for all study infants before SO was stopped and remained constant for Group 2 infants after SO was stopped. However, Group 1 infants had a significant decrease in the rate of weight gain (17.3 ± 13.1 vs 3.7 ± 6.1 g/kg/day), and the mean z scores for weight gain and weight for height also decreased significantly for Group 1 infants. Energy intake, incidence of acute infection, hematocrit values, and medication use did not differ before or after stopping SO in either group. CONCLUSIONS. This study indicated that short-term, awake S_{aO_2} measurements do not predict prolonged sleep S_{aO_2}, and overall, infants with BPD continued a positive growth trend when S_{aO_2} remained > 92% during prolonged sleep.

Maternal Smoking and Pulmonary Neuroendocrine Cells in Sudden Infant Death Syndrome—E Cutz, DG Perrin, R Hackman, EN Czegledy-Nagy. Pediatrics 1996;98(4):668.

BACKGROUND: Maternal smoking is a well-recognized risk factor for sudden infant death syndrome (SIDS), but the precise mechanism is unknown. We tested a hypothesis that maternal smoking affects pulmonary neuroendocrine cells (PNECs) and neuroepithelial bodies (NEBs), which are innervated PNEC clusters and presumed airway chemoreceptors. METHODS: Lung sections from infants who died of SIDS and whose mothers smoked during pregnancy (n = 22), infants who died of SIDS and whose mothers were nonsmokers (n = 17), and age-matched control infants (n = 15) who died of other causes were immunostained for bombesin (a PNEC and NEB marker) and assessed morphometrically. RESULTS: The frequency of PNEC (the percentage of airway epithelium immunoreactive for bombesin) was increased up to twofold in the lungs of infants who died of SIDS (7.7 ± 0.4%) compared with controls (4.9 ± 0.4%), as was the frequency (40 ± 3.5 vs 23 ± 3.7/cm^2) and size (748 ± 46.5 vs 491 ± 25.8 μm^2) of NEBs. In infants who died of SIDS and who were born to smoking mothers, PNEC frequency was increased significantly compared with that in those born to nonsmoking mothers, but the frequency and size of NEBs were not significantly different between the 2 groups. CONCLUSION: Our findings suggest that maternal smoking potentiates hyperplasia of the PNEC system in the lungs of infants who die of SIDS and that a dysfunction of these cells may contribute to the pathophysiology of SIDS.

Nicotine Patch Therapy in Adolescent Smokers—TA Smith, RF House Jr, IT Croghan, TR Gauvin, RC Colligan, KP Offord, et al. Pediatrics 1996;98(4):659.

OBJECTIVE: To evaluate the safety, tolerance, and efficacy of 24-hour nicotine patch therapy in adolescent smokers who were trying to stop smoking. DESIGN: Nonrandomized, open-label, 6-month clinical trial. SETTING: Five public high schools in the Rochester MN area. SUBJECTS: Twenty-two adolescent smokers, aged 13 through 17 years, with current smoking rate of 20 or more cigarettes per day (cpd). INTERVENTION: Daily nicotine patch therapy for 8 weeks (22 mg/d for 6 weeks followed by 11 mg/d for 2 weeks). Weekly individual behavioral counseling and group support continued for 8 weeks with follow up visits at 3 and 6 months and a mailed survey at 1 year. MAIN OUTCOME MEASURES: Self-reported smoking abstinence verified by expired air carbon monoxide of 8 ppm or less, nicotine withdrawal symptoms, adverse experiences and blood cotinine levels. RESULTS: Subjects had a mean ± SD smoking rate of 23.3 ± 5.0 (range, 20 to 35) cpd at study entry and 2.6 ± 1.6 years of smoking; the mean age was 15.9 ± 1.2 (range 13 through 17) years, and 68% were girls. Of the 22 participants, 19 (86%) completed patch therapy, 3 (14%) had biochemically validated smoking cessation at week 8, and 1 continued to be smoke free at 3 and 6 months after patch initiation. There was a significant decrease from baseline in the mean nicotine withdrawal scores for days 4 and 7 of Week 1 and the mean for Weeks 2 through 8. Skin reactions were the most common adverse event. As the worst skin reactions, 55% had erythema only, 5% had erythema and edema, and 9% had erythema and vesicles, whereas 32% had no skin reactions. Other reported adverse events were headaches (41%), nausea and vomiting (41%), tiredness (41%), dizziness (27%), and arm pain (23%). None of these were considered serious, life threatening, or led to the discontinuation of patch therapy. In adults with comparable smoking rates, we found that the adolescents had lower blood cotinine levels. Those smoking 20 to 25 cpd had cotinine levels of 146 ± 84 (adolescents) versus 260 ± 98 (adults) ng/mL, and those smoking 26 to 35 cpd had levels of 169 ± 73 versus 276 ± 110 ng/mL, respectively. CONCLUSION: Nicotine patch therapy seems safe in adolescent smokers. Placebo-controlled trials are needed to establish the efficacy of nicotine patch therapy in adolescents.

Comparison of Regularly Scheduled with As-Needed Use of Albuterol in Mild Asthma—JM Drazen, E Israel, HA Boushey, VM Chinchilli, JV Fahy, JE Fish, et al. N Engl J Med 1996;335 (12):841.

BACKGROUND: Inhaled β-agonists are the most commonly used treatment for asthma, but data suggest that regularly scheduled use of these agents may have a deleterious effect on the control of asthma. We compared the effects of regularly scheduled use of inhaled albuterol with those of albuterol used only as needed in patients with mild chronic, stable asthma. METHODS: In a multicenter, double-blind study, we randomly assigned 255 patients with mild asthma to inhale albuterol either on a regular schedule (126 patients) or only as needed (129 patients). The patients were followed for 16 weeks. RESULTS: The primary outcome indicator, peak expiratory air flow measured in the morning, did not change significantly during the treatment period in the scheduled (416 L/min after the run-in period and 414 L/min after the treatment period) or the as-needed (424 L/min at both times) treatment groups (p = 0.71). There were no significant differences between the 2 groups in peak flow variability, forced expiratory volume in 1 second, the number of puffs of supplemental albuterol needed, asthma symptoms, asthma quality-of-life score, or airway responsiveness to methacholine. The statistically sig-

nificant differences between the groups in evening peak flow and in the short-term bronchodilator response to inhaled albuterol were small and judged to be clinically unimportant. CONCLUSIONS: In patients with mild asthma, neither deleterious nor beneficial effects derived from the regular use of inhaled albuterol beyond those derived from use of the drug as needed. Inhaled albuterol should be prescribed for patients with mild asthma on an as-needed basis. *See the related editorial:* ***Inhaled β_2-Agonists in the Treatment of Asthma.*** *PM O'Byrne, HAM Kerstjens. N Engl J Med 1996; 335(12):886-888.*

Diaphragm Strength in Chronic Obstructive Pulmonary Disease—MI Polkey, D Kyroussis, C-H Hamnegard, GH Mills, M Green, J Moxham. Am J Respir Crit Care Med 1996;154:1310.

The diaphragm is normally the main inspiratory muscle, and diaphragm strength in chronic obstructive pulmonary disease (COPD) is therefore of interest. We assessed diaphragm strength in 20 patients with severe stable COPD (mean FEV_1 0.61, mean thoracic gas volume [Vtg] 5.31) and 7 normal control subjects, measuring both maximal sniff transdiaphragmatic pressure (sniff Pdi_{max}) and twitch transdiaphragmatic pressure (Tw Pdi) elicited by cervical magnetic stimulation (CMS) of the phrenic nerve roots at FRC. Acute-on-chronic hyperinflation was examined in 4 patients. Mean Tw Pdi in patients and control subjects was 18.5 cm H_2O and 25.4 cm H_2O, respectively ($p < 0.01$), and mean sniff Pdi was 81.9 cm H_2O and 118 cm H_2O, respectively ($p < 0.001$). Reduction in mean intrathoracic pressures was more marked; twitch esophageal pressure (Tw P_{es}) was −7.3 cm H_2O and −16.3 cm H_2O, respectively ($p < 0.001$) and sniff P_{es} was −67 cm H_2O and −97.8 cm H_2O ($p < 0.001$). During acute-on-chronic hyperinflation there was a linear negative correlation of Tw Pdi with increasing lung volume of 3.5 cm H_2O/L. The ability of the diaphragm to generate transdiaphragmatic, and particularly a negative intrathoracic, pressure is reduced in COPD and these changes are exaggerated with acute-on-chronic hyperinflation.

Risk Factors for Pneumonia, and Colonization of Respiratory Tract and Stomach in Mechanically Ventilated ICU Patients—MJM Bonten, DCJJ Bergmans, AW Ambergen, PW de Leeuw, S van der Geest, EE Stobberingh, CA Gaillard. Am J Respir Crit Care Med 1996;154:1339.

Risk factors for the development of ventilator-associated pneumonia (VAP) and colonization of the respiratory tract and stomach with enteric Gram-negative bacteria (EGB) and Pseudomonadaceae were determined in 141 ventilated patients using univariate analysis and the Cox proportional hazards model. VAP was caused by EGB in 14 patients (10%), and by Pseudomonadaceae in 19 patients (13%). The duration of ventilation was a significant risk factor for VAP caused by EGB and Pseudomonadaceae, and for acquired colonization in oropharynx, stomach, and trachea with these species. Of 20 other variables, oropharyngeal colonization with EGB on admission (hazard ratio [HR] = 4.5) and an infection on admission (HR = 2.7) were selected as risk favors for VAP caused by EGB. Acquired colonization with Pseudomonadaceae in oropharynx (HR = 5.0) was the most important risk factor for VAP caused by these species. Gastric colonization with EGB or Pseudomonadaceae were no risk factors for VAP. For acquired oropharyngeal colonization with EGB only the duration of ventilation was a risk factor, whereas preceding colonization of the trachea with Pseudomonadaceae and duration of ventilation were risk factors for acquired oropharyngeal colonization with these species. In the Cox model, only the duration of ventilation was significantly related to acquired gastric colonization with EGB. Preceding colonization of the oropharynx and of the trachea with Pseudomonadaceae were risk factors for acquired colonization with these species in the stom-

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ach. Twelve patients with VAP (46%) and 38 without VAP (33%) died ($p = 0.21$). In conclusion, duration of ventilation and colonization of the upper respiratory tract are the most important risk favors for VAP caused by EGB or Pseudomonadaceae.

Efficacy of Cardiopulmonary Resuscitation Using Intratracheal Insufflation—L Brochard, G Boussignac, S Adnot, C Bertrand, D Isabey, A Harf. Am J Respir Crit Care Med 1996;154:1323.

The effects of constant-flow insufflation (CFI) of air in the trachea at the distal end of a modified endotracheal tube as the sole mode of ventilation during cardiopulmonary resuscitation (CPR) were studied in pigs. The ventilatory effect of CFI (15 ± 2 L/min) generating a positive pressure of about 10 cm H_2O with concomitant chest compression was studied first. In 9 sedated, paralyzed animals disconnected from the ventilator, CFI alone did not significantly alter the decrease in $P_{a(O_2}$ and the rise in $P_{a(CO_2}$ observed during apnea. By contrast, the combination of precordial compression and CFI (CFI-CPR) maintained arterial blood gases over a 4-minute period at the level obtained during mechanical ventilation. In the second part of the study, ventricular fibrillation was induced and CFI-CPR was compared with standard CPR using conventional mechanical ventilation during 2 successive 4-minute periods, in random order. Ventilatory parameters were identical in the 2 situations, whereas hemodynamic parameters were similar or better with CFI-CPR than with standard CPR. Significant differences were observed between standard CPR and CFI-CPR for systolic aortic pressure (72 ± 22 vs 82 ± 27 mm Hg, respectively; $p < 0.02$) and for systolic (322 ± 216 vs 431 ± 237 mL/s; $p < 0.01$) and mean (116 ± 106 vs 143 ± 108 mL/s; $p < 0.01$) common carotid blood flows. The ease of use of CFI together with its beneficial hemodynamic effects suggests that CFI deserves to be investigated further as a mode of ventilation during CPR.

Descriptors of Breathlessness in Cardiorespiratory Diseases—DA Mahler, A Harver, T Lentine, JA Scott, K Beck, RM Schwartzstein. Am J Respir Crit Care Med 1996;154:1357.

The purposes of this study were: to examine the descriptors of breathlessness chosen by a large sample of patients with cardiorespiratory disease; to determine test-retest reliability of a patient's selection of the descriptors; and to assess whether a patient's recall of the experience of breathlessness is the same as that provoked by physical activity. Questionnaire data were collected at an initial visit for patients who complained of breathlessness and at a second visit in a subgroup of patients. A total of 218 patients who sought medical care for difficulty breathing due to 1 of 7 different conditions were recruited from an outpatient pulmonary disease clinic at a university medical center. Patients selected statements that described qualities of breathlessness from a 15-item questionnaire and completed pulmonary function tests. At a subsequent visit (4 to 15 days later) a subgroup of 16 patients with chronic obstructive pulmonary disease (COPD) repeated the questionnaire at rest (to assess reliability) and after walking in a hallway to provoke a moderate intensity of breathlessness (to compare recall with direct experiences). The relationship among descriptors was evaluated by cluster analysis. The "work/effort" cluster was common for all diagnoses. Each condition was characterized by more than 1 cluster except COPD. Each diagnosis was associated with a unique set of clusters (eg, asthma with "work/effort" and "tight," interstitial lung disease with "work/effort" and "rapid" breathing). Percent agreement for all descriptors selected at Visits 1 and 2 (recall) was 79% ($r = 0.82$; $p = 0.001$). Percent agreement at Visit 2 between descriptors for recall and for breathlessness provoked by walking was 68% ($r = 0.69$; $p = 0.004$). We conclude that patients with different cardiorespiratory conditions experience distinct qualities of breathlessness. Patients' recall of their sensations of breathlessness is reliable and comparable to dyspnea with walking. Employing a questionnaire containing descriptors of breathlessness may help to establish a specific diagnosis and to identify mechanisms whereby a specific intervention relieves dyspnea.

Does Dietary Intake of Vitamins C and E Influence Lung Function in Older People?—L Dow, M Tracey, A Villar, D Coggon, BM Margetts, MJ Campbell, ST Holgate. Am J Respir Crit Care Med 1996;154:1401.

Antioxidants in the lung have a protective role against oxidative damage. We have investigated whether dietary antioxidant intake in elderly people is related to lung function. Dietary intakes of ascorbic acid (vitamin C) and alpha-tocopherol (vitamin E) and lung function were assessed in 178 men and women aged 70 to 96 years selected on the basis of reported respiratory symptoms. After adjustment for age, gender, height, smoking habits, total energy intake, and vitamin C intake by multiple linear regression, vitamin E intake was significantly associated with FEV_1 $p = 0.012$ and FVC $p = 0.003$. There was no evidence of any interaction between vitamin E intake and current tobacco smoking as determinants of lung function. For every extra milligram increase in vitamin E in the daily diet, FEV_1 increased by an estimated 42 mL and FVC by an estimated 54 mL. These results suggest that dietary intake of vitamin E may influence lung function in the elderly, but food frequency questionnaires in this study were not of sufficient sensitivity to explore this hypothesis further.

Chest X-Ray Changes in Air Space Disease Are Associated with Parameters of Mechanical Ventilation in ICU Patients—EW Ely, MM Johnson, C Chiles, JT Rushing, DL Bowton, RI Freimanis, RH Choplin, EF Haponik. Am J Respir Crit Care Med 1996;154:1543.

To assess relationships between parameters of mechanical ventilation (MV) and portable chest X-ray (CXR) measurements of lung length (LL) and severity of air space disease, a prospective, randomized, blinded comparison of 102 adults in a university hospital was performed. Each patient received 2 portable, supine CXRs on different MV breaths within 5 minutes of one another. Ventilator parameters were recorded. All 204 CXRs were randomly assorted and read independently by 3 radiologists. Air space disease was considered more severe with pressure support ventilation (PSV) breaths than with intermittent mandatory ventilation (IMV) breaths ($p = 0.0003$), and its extent correlated inversely with static compliance ($p = 0.0001$, $r = -0.40$). Among patients having CXRs on both IMV and PSV breaths, 15 of 67 (22%) had their overall degree of air space disease read differently by 1 category (mild, moderate, or severe). Increases in LL between the 2 CXRs were associated with increasing peak ($p = 0.0038$) or mean ($p = 0.0065$) airway pressure, tidal volume (V_T) ($p = 0.022$), and V_T per kilogram ($p = 0.006$). We conclude that lung volume changes during MV, typically not noted nor controlled for during portable chest radiography, may substantially alter the interpretation of air space disease and LL. Physicians monitoring intensive care unit (ICU) patients with daily CXRs should be aware of the variables influencing interpretation of portable CXRs of ICU patients.

Use of a Pleural Catheter for the Management of Simple Pneumothorax—T Martin, G Fontana, J Olak, M Ferguson. Chest 1996;110(5):1169.

OBJECTIVE: We assessed the use of a pleural catheter (Thoracic Vent) to determine its effectiveness in treating simple pneumothorax (PTX) and in preventing recurrent PTX. DESIGN: A retrospective review was conducted of 84 patients treated with a pleural catheter for iatrogenic (52) and spontaneous (11 primary, 21 secondary) PTX between 1989 and 1994. PATIENTS: There were 45 men and 39 women with a mean age of 50.4 years (range, 18 to 85 years). RESULTS: Mean time to lung re-expansion was 0.5 ± 0.1 days. Forty-five (57%) patients manifested an air leak after catheter placement for 2.0 ± 0.2 days. The duration of time to catheter removal was 3.3 ± 0.2 days. Seventy-one (85%) patients had resolution of PTX with this therapy alone. Thirteen patients (15%) failed to resolve their PTXs and required subsequent tube thoracostomy alone (6) or surgical therapy (7). Four of 11 patients who required tube thoracostomy also failed to respond to this therapy. Treatment failure was more common among patients with spontaneous PTX than with iatrogenic PTX (34% vs 4%; $p < 0.005$). During a mean follow-up of 3.0 ± 0.2 years, 6 (7%)

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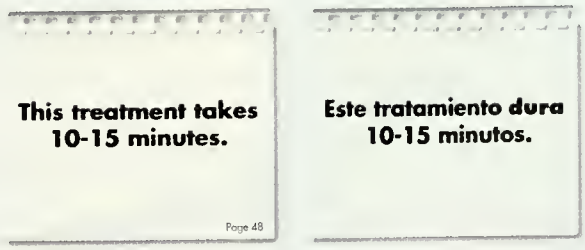

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patients suffered recurrent PTX an average of 23 days after initial therapy. CONCLUSION: This pleural catheter is effective in the management of simple iatrogenic and spontaneous PTX.

Bronchodilatation and the Inspiratory Flow Volume Curve—R Reddy, T Cook, MF Tenholder. Chest 1996;110(5):1226.

The inspiratory flow volume curve is included in the spirometric tracing provided by most pulmonary function laboratories and is useful in assessing upper airway abnormalities. We analyzed the changes in peak inspiratory flow (PIF), forced inspiratory flow at 50% (FIF_{50}), and forced inspiratory vital capacity (FIVC) that occur with bronchodilator challenge testing (3 puffs of a β-agonist by metered dose inhaler without a spacer device) in 145 consecutive patients. Fifty-five patients responded to bronchodilators with either a 12% or 200-mL change in FEV_1 or FVC. All of these subjects produced forced expiratory maneuvers that fulfilled American Thoracic Society criteria or acceptability. The FIVC was the only inspiratory parameter that consistently showed a similar correlation to the bronchodilator responsiveness demonstrated by the FEV_1 or FVC. With the reduction in air trapping, a 12% or 200-mL change in the FIVC is additional confirmatory evidence of bronchodilator responsiveness.

Viability, Morbidity, and Resource Use among Newborns of 501- to 800-g Birth Weight—JE Tyson, N Younes, J Verter, LL Wright, for the National Institute of Child Health and Human Development Neonatal Research Network. JAMA 1996;276(20):1645.

OBJECTIVES: To assess risk factors affecting viability and analyze the effects of mechanical ventilation (MV) on neonatal outcome and resource use among extremely premature infants. DESIGN: Inception cohort study. SETTING: Neonatal intensive care units of the 12-center National Institute of Child Health and Human Development Neonatal Research Network. PARTICIPANTS: A total of 1,126 infants with a birthweight of 501 to 800 g born in network centers between January 1, 1994, and December 31, 1995. MAIN OUTCOME MEASURES: Observed survival; maximum estimated survival (assuming the same survival among infants who died without MV as among infants in the same risk category who received MV); observed and maximum estimated survival without severe brain injury (either interventricular echodensity with ventricular dilation or parenchymal echodensity); hospital stay; resource investment. RESULTS: Overall mortality was 43%; mortality in infants without MV was 93%. A total of 15% of all the infants died without MV. Females, small-for-gestational-age infants, and infants whose mothers received antenatal steroids had an advantage in survival with MV equivalent to an increase in birthweight of 90 g, 57 g, and 67 g, respectively. The corresponding advantage of these infants in survival without severe brain injury was 107 g, 97 g, and 64 g, respectively. Females in the lowest birthweight group were more likely to die without MV than were larger males with a similar estimated likelihood of survival with MV. Mean hospital stay was 115 days for the survivors, values much greater than the 17.9-day standard for 501- to 800-g survivors under the diagnosis-related group system. Resource investment was considerable (127 hospital days/survivor and 148 days/survivor without severe brain injury), but, like outcome, varied markedly between risk categories. Had MV been used for all infants who died, we estimate a substantial increase in resource use and a maximum of 8 additional survivors (no more than 6

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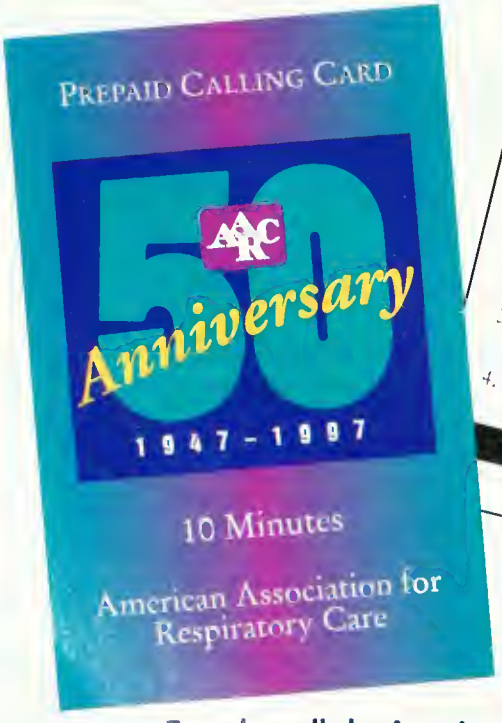

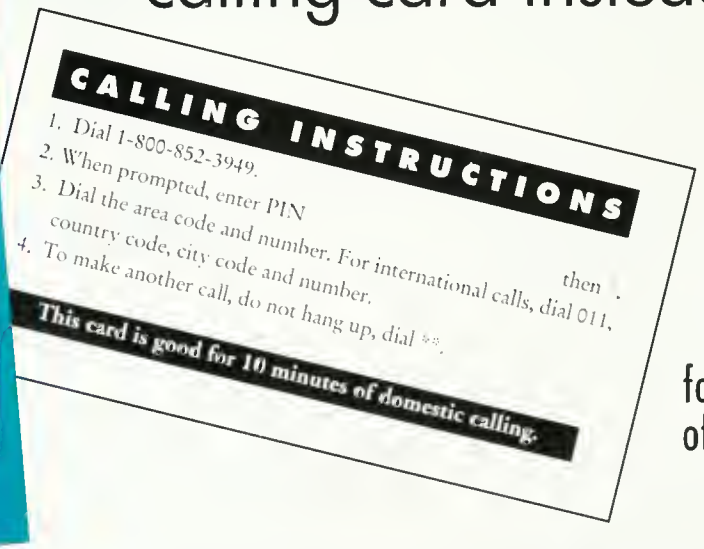
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without severe brain injury per 100 infants with a birthweight of 501 to 800 g. CONCLUSIONS: Although recommendations to initiate or forgo MV for extremely premature infants have often focused on 1 factor (birthweight or gestational age), multiple factors should be considered. Other factors being equal, our analyses support use of MV for females at a minimum birthweight approximately 100 g lower than that for males. The current diagnosis-related group reimbursement system can be expected to compromise resources for 501- to 800-g infants who would benefit from MV. Such care entails considerable resource use, although the cost per life-year gained is likely to be considerably less than that for many adults given intensive care. Our findings can be used to facilitate more appropriate treatment decisions, determine adequate resources, and better inform the debate about the benefits and burdens of intensive care for extremely premature newborns.

Comparison of the Effects of Heat and Moisture Exchangers and Heated Humidifiers on Ventilation and Gas Exchange During Weaning Trials from Mechanical Ventilation—G Le Bourdellès, L Mier, B Fiquet, K Djedaïni, G Saumon, F Coste, D Dreyfuss. Chest 1996;110(5):1294.

Heat and moisture exchangers (HME) are increasingly used to warm and humidify inspired gases in intubated ventilated patients. But these devices add dead space that may alter the alveolar ventilation. This could impair the efficiency of spontaneous ventilation (SV) during weaning trials from mechanical ventilation. Fifteen patients were tested with an HME (Hygrobac-DAR) and a heated humidifier (HH) (Fischer-Paykel MR 450) in a random order during weaning trials in SV with inspiratory pressure support. Minute ventilation ($\dot{V}_E$, tidal volume), and respiratory rate were recorded and arterial blood was sampled for blood gas analysis with each device. The HME gave a significantly greater $\dot{V}_E$ than the HH (9.3 ± 0.8 L/min vs 8.1 ± 0.8 L/min; $p < 0.005$), because of increased respiratory rate (21 ± 2/min vs 19 ± 2/min; $p < 0.05$). Tidal volume was unchanged for HME and HH (470 ± 32 mL vs 458 ± 39 mL). The higher P_{aCO_2} with HME than with HH (44 ± 2 mm Hg vs 42 ± 2 mm Hg; $p < 0.005$) revealed an insufficient alveolar ventilation response to the increase in dead space. Arterial P_{O_2} rose with the HME, but not significantly above the HH values (103 ± 6 mm Hg vs 97 ± 6 mm Hg; $p = 0.055$), possibly because of a positive end-expiratory pressure effect of the HME. The need to increase $\dot{V}_E$ in SV when an HME is used should be taken into account during difficult weaning from mechanical ventilation.

Variations in the Use of Do-Not-Resuscitate Orders in ICUs: Findings from a National Study—RL Jayes, JE Zimmerman, DP Wagner, WA Knaus. Chest 1996;110(5):1332.

OBJECTIVES: To describe the variation in frequency of do-not-resuscitate (DNR) orders in 42 U.S. ICUs and to examine the relationship between published guidelines and qualitative observations about terminal care in 9 ICUs. DESIGN: Prospective inception cohort. SETTING: Forty-two ICUs in 40 U.S. hospitals with more than 200 beds; 26 randomly selected and 14 large tertiary-care volunteers. PARTICIPANTS: A consecutive sample of 17,440 ICU admissions during 1988 to 1990. MEASUREMENTS & RESULTS: We used age, race, comorbid conditions, disease, functional status, and acute physiology score on ICU Day 1 to predict the likelihood of a DNR order for each patient. A cross-validated model was then used to predict variations in the risk of an ICU DNR order from 0 to 45% (area under receiver operating characteristic curve = 0.9). The model was then used to compare aggregate observed with predicted frequency of ICU DNR orders. Finally, we compared observations of DNR practices by a team of clinical and organizational researchers at 9 of the 42 ICUs with published guidelines and risk-adjusted DNR frequency: 1,577 admissions (9%) had DNR orders written in the ICU (range, 1.5 to 22%). The ICU site was a significant ($p < 0.0001$) predictor of variance in the patient level model. DNR orders were written significantly ($p < 0.05$) less frequently than predicted in 5 and more frequently than predicted in 3 of 42 ICUs. Nonwhite patients had significantly ($p = 0.0001$) fewer DNR orders after adjustment. The research team's implicit judgments following on-site analysis failed to distinguish ICUs with more or less DNR orders than predicted. Site-visited ICUs exhibited practices to emulate and practices to avoid. CONCLUSIONS: The frequency of ICU DNR orders can be predicted based on individual risk factors for groups of ICU patients. After adjusting for differences in patient characteristics, there is significant variation in the frequency of DNR orders in a national sample of ICUs. These variations may be due to unmeasured differences in patient characteristics such as treatment preferences, religious affiliation, educational level, or physician practices. We found no relationship between risk-adjusted DNR order frequency and adherence to published guidelines. *See the related editorial:* ***Variations in DNR Rates: The Onus Is on Physicians****—TJ Prendergast, TA Raffin. Chest 1996; 110(5):1141-1142.*

RESPIRATORY CARE

April 1997
Special Issue

Noninvasive Positive Pressure Ventilation based on the Consensus Conference held October 4-6, 1996, in Vail, Colorado. Sponsored by the Journal and the American Respiratory Care Foundation

Philip Kittredge RRT

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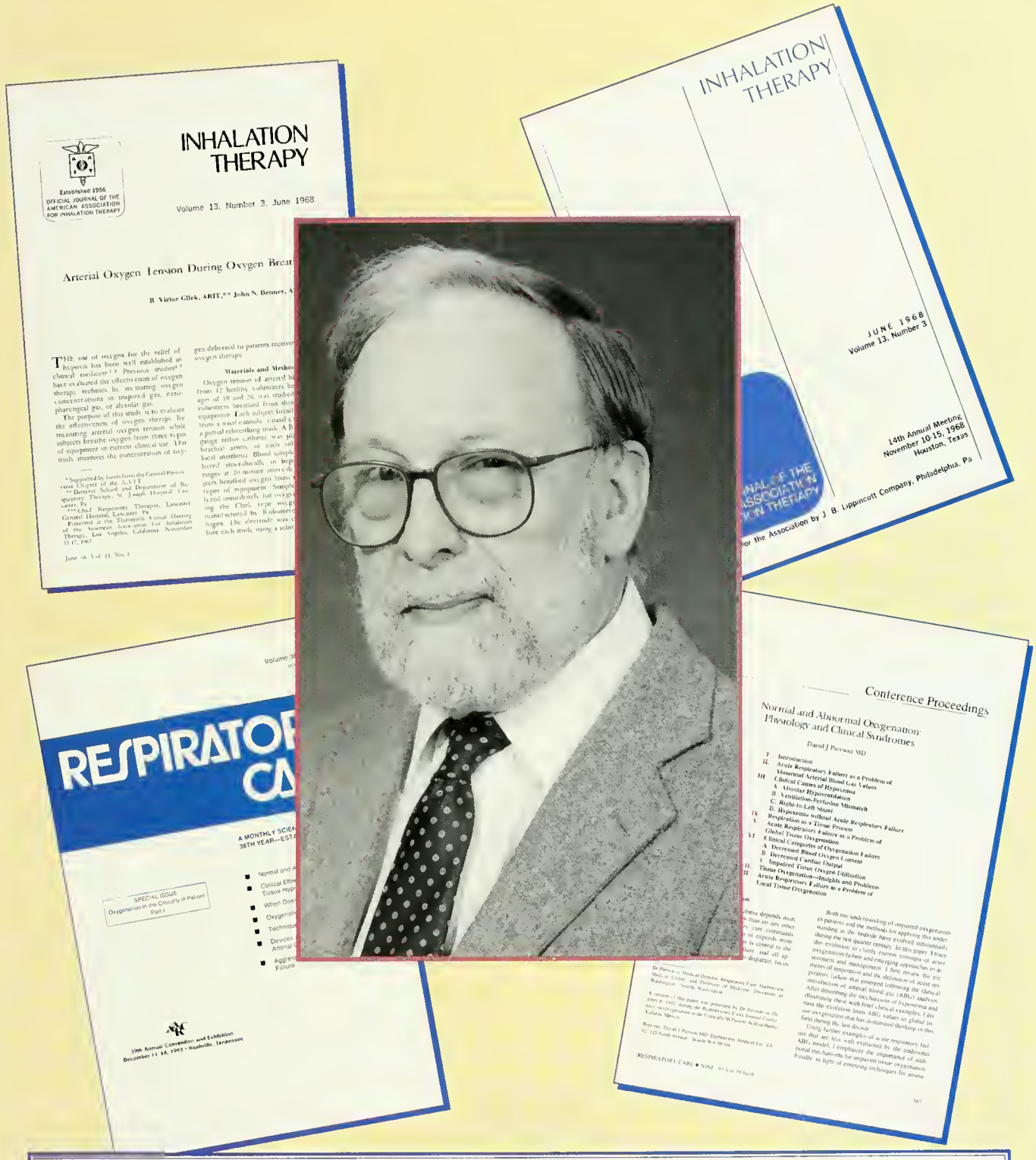

Philip Kittredge died a few days before Christmas 1996, following an extended period of declining health. Among the many who have contributed substance and integrity to the respiratory care profession, Phil stood at the forefront. His vision, constancy, and unwavering commitment are unparalleled. Without that vision, constancy, and commitment, there would be no Journal, and there might be little scientific foundation for respiratory care. Phil's contribution spanned 31 years—25 of those as Editor of RESPIRATORY CARE.

Capnography—A Key to Understanding Physiology

Capnography is the measurement and graphic display of the carbon dioxide (CO_2) concentration in the respiratory gas mixture, measured throughout the respiratory cycle. The technology for capnography has been available for more than 50 years.[1] Over the past 2 decades, interest in the clinical utilization of this technology has been increasing. Specific interest has centered around the use of the so-called end-tidal carbon dioxide tension (P_{etCO_2}) as a substitute for measurement of arterial carbon dioxide tension (P_{aCO_2}). In normal healthy individuals, it is reasonable to believe that the P_{etCO_2} (an estimate of alveolar CO_2) would closely approximate P_{aCO_2}. Almost 15 years ago, this clinical application in critically ill patients was called into question,[2,3] and widespread discussion continues today concerning the clinical utilization of capnography in the critical care setting.

Several recent studies[4-7] have called into question the reliability of the relationship between P_{etCO_2} and P_{aCO_2} in patients receiving mechanical ventilatory assistance. This should be expected as the P_{etCO_2} represents the tension of CO_2 being removed from the lungs, and the P_{aCO_2} represents the amount of CO_2 remaining in the blood. It would seem that the difference between the P_{etCO_2} and the P_{aCO_2} is directly related to the lung's ability to remove CO_2 from the body, but this remains a controversial subject, and active investigation is ongoing to more clearly define this relationship.

Several accepted clinical applications of capnography exist. They are discussed in an AARC Clinical Practice Guideline[8] and include determination of tracheal versus esophageal intubation, continuous monitoring of the integrity of the ventilatory circuit, evaluation of efficiency of mechanical ventilation support reflecting CO_2 elimination, monitoring adequacy of pulmonary and coronary blood flow, monitoring concentration of inspired CO_2, monitoring the severity of pulmonary disease, and graphic evaluation of the ventilator-patient interface.

We have the technology to measure the CO_2 concentration in the respiratory gases. We also know this determination should not replace the analysis of arterial blood to determine the P_{aCO_2}. As smart clinicians and physiologists, we need to now recognize the importance of the capnograph, especially the analysis of the capnographic waveform and the relationship between the exhaled CO_2 and the arterial CO_2 tensions. Three aspects of recent research and publication need to be discussed.

First, the value of the capnograph in detecting 'mishaps.' Little information is available in the critical care literature concerning this. However, the anesthesia literature has several reports of large series of cases evaluating the clinical utility of various monitoring technologies, including capnography, for detecting mishap events during anesthesia. Three large series, one from the United States,[9] one from Australia and New Zealand,[10] and the third from Scandinavia[11] have all concluded that the presence of capnographic monitoring is the second most useful monitoring modality for detecting mishap events, with pulse oximetry being first and electrocardiographic monitoring a distant third. These reports have led to the implementation worldwide of capnographic monitoring as part of the standard of care during anesthesia.[12]

Second, reports analyzing the capnographic waveform, with particular interest being given to the slope of Phase II (the rapidly increasing phase) and the angle between Phase II and Phase III (the plateau phase) have been published. Monitoring the slope of Phase II has been suggested as a useful and reliable monitor of the progression of lung pathology during the acute respiratory distress syndrome.[13,14]

SEE THE RELATED CASE REPORT ON PAGE 218 AND THE GRAPHICS CORNER ON PAGE 227.

Third, recent interest has centered on the prognostic value of various monitoring tools. A recent report evaluated the usefulness of capnography and related derived values (such as the P_{etCO_2}, $P_{aCO_2} - P_{etCO_2}$, and estimated dead space-to-tidal volume ratio, or V_D/V_T) as prognostic tools of poor outcome during anesthesia care.[15] The authors report on 100 critically ill or injured, trauma or major surgery patients having an overall mortality of 41%. In this series of patients the estimated V_D/V_T [$(P_{aCO_2} - P_{etCO_2})/P_{aCO_2}$] had significant prognostic value. For patients in whom the estimated V_D/V_T was > 65% (n = 10) at any time during the surgery, there was 100% mortality. For patients in whom V_D/V_T remained < 20% (n = 14) during the surgical procedure the mortality was 7%. For the group of patients with V_D/V_T between 20 and 65% (n = 76) the mortality was 39%. Although these data evaluate intraoperative monitoring and come from a relatively small population, I believe that they strongly suggest the prognostic value of the relationship between P_{etCO_2} and P_{aCO_2}. Further investigation in the critical care setting remain to be done.

Two articles appearing in this issue of RESPIRATORY CARE[16,17] highlight the clinical use of capnography for providing important information about a patient's clinical condition. Both cases describe common, relatively 'simple' clinical situations (laparoscopic cholecystectomy) and the use of capnography to provide important information concerning the patient's ongoing clinical condition and the underlying relevant physiology.

Should the use of capnography in the critical care setting be discontinued because of the lack of correlation between the P_{aCO_2} and the P_{etCO_2}? Of course not, any good respiratory physiologist (which all respiratory care professionals should be) already understands that only in normal healthy individuals can the P_{aCO_2} and P_{etCO_2} be expected to correlate reasonably well. If we realize the clinical importance of determining the relationship between the P_{etCO_2} and P_{aCO_2} (evaluating the ability of the lung to act as an organ of excretion, which it is) and of analyzing the capnographic waveform, then we can use capnography appropriately in the critical care setting.

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Benchmarking Applied to Health Care

Managed competition, rising consumer expectations, and a restructuring of the reimbursement system are placing enormous demands on hospitals to improve quality and maximize operational efficiencies. Respiratory care practitioners are being called upon to introduce breakthroughs in performance at a record pace to meet the new and dynamic requirements of the marketplace. In an effort to discover these breakthroughs, department directors have incorporated benchmarking into their strategic process.

Many of the basic ideas on which benchmarking is founded have been around for several years. The Japanese word *dantotsu*, which means striving to be the best of the best, captures the essence of benchmarking. Benchmarking is most often defined as "the continuous process of measuring products, services, and practices against one's toughest competitors or a renowned industry leader."[1]

SEE THE RELATED EDITORIAL ON PAGE 204 AND THE COMMENTARY ON PAGE 222.

Three types of benchmarking are generally recognized: collaborative, functional, and internal. Collaborative benchmarking enables an organization to learn from the best practices within a voluntary network of health care providers (eg, Voluntary Hospitals of America or University Health Systems Consortium). Collaborative benchmarking is often managed by professional organizations.

Functional benchmarking compares a work function with the functional leader even when the leader is in a different industry. However, clinical functions, by their technical nature, restrict the search for benchmarking partners to health care organizations.

Internal benchmarking involves the identification of best practice within one's organization. Internal benchmarking is both a free-standing exercise to improve existing performance and a low-risk way to share performance data. By publishing performance data, others can learn what a high-performing organization is doing to achieve results. In this issue of RESPIRATORY CARE, Chatburn et al[2] present internal benchmarking data on the outcomes of mechanical ventilation in pediatric patients.

Benchmarking depends on the disciplined collection and use of objective information.

The paradigm is simple and entails

- Identifying critical success factors and defining key indicators;
- Collecting information relevant to the key indicators;
- Searching to identify extraordinary performers, as defined by the data points;
- Identifying the factors that drive superior performance;
- Adopting or adapting those factors that fit into your processes.

Benchmarking indicators are of three basic types:

Ratio Indicators—Indicators that establish a relationship between two measures (example: worked hours/unit of service). Ratio indicators are generally indicative of productivity or of a volume measurement. They provide a comparative performance point to other departments and/or hospitals, but do not reveal information about the practices that drive the performance.

Process Indicators—Indicators that measure a process with a beginning point and an ending point (example: blood gas turn-around time; time from blood drawn to results in patient's chart). Process indicators lead to investigations of the practice that drives the performance.

Outcome Indicators—Indicators that measure clinical outcomes (example: patient returns to the emergency department within 24 hours). Outcome indicators lead to an understanding of the practices that provide the best possible clinical outcomes.

Once the key indicators have been identified useful information (data) about existing processes are collected.

Data Collection

Most quality improvement tools depend on reliable, accurate data. Without data, efforts are reduced to a 'guessing game' with relatively low odds of success. Checklists are used to gather information (answers to questions) while data sheets are seeking data (facts).

A checklist is a simple information-recording form that has been specifically designed to provide descriptive information. When one attempts to compare measures from one department to another, the comparison is meaningless unless

the measures are defined in such a way that we are comparing 'apples to apples.'

Data sheets differ from checklists in that the data are recorded in a simple tabular or columnar format. The form is specifically designed to obtain bits of data, usually numbers or predefined words, that are indicative of a value or an event.

Gap Analysis

Just as the name implies, this analysis is designed to identify gaps among the benchmarking participants. One format to present the data employs statistically derived comparisons using rates and ratios. The aim of this type of data is to determine whether the value of a given variable differs from a like value of similar populations. These are called tests of significance. A second format, and, perhaps, more useful to most, is visual representation in the form of charts and graphs. The most common is the bar chart. A bar chart is particularly useful when one wishes to display and compare relative magnitudes of the measurements for each of the categories under consideration.

Once the gap analysis is complete and results are known, individuals are likely to respond in one of three ways: denial, rationalization, or learning.

Seldom will the results of a benchmarking project proclaim any department and/or hospital 'best-of-class' across the board. More often the news is less than uplifting, and, perhaps, even discouraging. The natural response from a manager is "These data can't be correct." Unfortunately, they probably are. 'Swallowing the bitter pill' of reality is the most challenging part of benchmarking.

The second response is rationalization. In the attempt to rationalize the gaps identified in the benchmarking process, managers usually try to find errors in the data and/or the methods used to collect the data. If an error can be uncovered, then business can continue as usual. The cry is often that "We're unique," and the implication is that just because a methodology worked in Hospital A doesn't mean that it will work for us because we are different. In my own situation I could say "I can see how an adult hospital could achieve efficiencies by consolidating services, but it's different in a pediatric hospital." In general, we try to explain away the differences.

It is fortunate that from humility comes an enthusiasm to learn. Learning is a third response. Learning comes from accepting reality and taking actions to change it. Corrective action begins with asking the right questions and accepting that the benchmarking data are probably correct and that lessons can be learned from our benchmarking partners. Collins and Huge[3] have called humility the Number 1 factor in learning: "Without humility, there will be no real learning and change." A remark often repeated by Ray Kroc, founder of MacDonald's, is "When you're green, you grow. When you're ripe, you rot."

The overriding objective of benchmarking is to identify and learn about best practices. But, unless we implement the best practices, we have engaged in nothing more than an intellectual exercise with little value. For benchmarking to have value, it must be put to work improving your organizational performance.

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Benchmarking: It's Time To Get on with It

Two pieces in this issue of the Journal[1,2] focus on benchmarking—defining the concept, providing some background, and describing one institution's foray into such activity. I want to complement those two pieces by further delineating what I believe to be the importance of benchmarking to the respiratory care profession today.

Our profession needs to pay serious attention to the benchmarking process. Health care organizational decision makers increasingly are using this tool to position their organizations as cost-effective, high-quality providers. The 1996 biennial Deloitte & Touche LLP survey,[3] which reflects the opinions of 1,020 hospital executives, indicated that 76% of the respondents view outcomes measurement and management as a key strategy for market success. This is up from 57% just 2 years ago. If our bosses think this is important, we ought to be paying attention.

As health care organizations lose the ability to differentiate themselves on the basis of price, they will (fortuitously enough for the consumer!) compete more intensely on the basis of quality. They will compare their performance to their competitors on indicators important to the marketplace. Purchasers of health care services will make contracting decisions based on the value (quality services at a competitive price) offered by each provider. They will define both cost and quality by indicators.

Several health care organizations have addressed the need for comparative outcome data. The National Committee for Quality Assurance (NCQA), an independent, not-for-profit organization whose mission is to provide information that enables purchasers and consumers of managed health care to distinguish among plans based on quality, has developed one such system. Their performance measurement system, HEDIS 3.0,[4] facilitates the comparison of health plans and motivates health plans to develop appropriate information systems and create internal quality improvement programs. The federal government, the largest payer of health care expenditures, has recognized the value in performance measurement. Beginning this year, managed care plans that serve Medicare beneficiaries will be required to submit data on those HEDIS measures relevant to Medicare. In the July/August 1996 issue of Joint Commission Perspectives,[5] Joint Commission for the Accreditation of Healthcare Organizations President Dennis O'Leary MD, reported that a proposal for performance measurement and improvement in accredited hospitals and long-term-care organizations will be considered by their Board of Commissioners. The proposal, if accepted, would require that accredited organizations select, by January 1, 1998, a performance measurement system and at least two clinical performance indicators that address at least 20% of their patient population. By the first quarter of 1999, they would require the submission of summary data on indicator performance.

SEE THE RELATED EDITORIAL ON PAGE 202 AND THE COMMENTARY ON PAGE 222.

Comparative cost data are equally important. The health care industry has long had performance indicators that enable executives to compare their financial performance and efficiency with other similar organizations. Our profession has not yet done this. We need to establish indicators that will enable each manager to know how his or her department's efficiency in service delivery and quality of care compares with others. Many consultants have used and will continue to use the lack of comparative data to assail the value of our departments during organizational restructuring. They tell administrators that respiratory care departments are inefficient, and they produce gross comparisons like procedure counts taken from financial reports to make their point. Their assumption of convenience propounds that all respiratory care procedures require the same time to complete. Thus, they perceive comparing procedures as an efficient method of comparing productivity. Variables that drive human resource demand like scope of service and the mix of procedures provided are critical in assuring fair comparisons between different respiratory care departments. Unfortunately, these are often not considered by busy executives who are distant from daily operations.

If establishing performance indicators and a comparative database for respiratory care services were easy to do, it would have been done by now. The truth is that it is an enormous undertaking. When comparing activities all participants must be using a uniform language or 'apples-to-apples' comparisons cannot be made. Although the American Association for Respiratory Care (AARC) Uniform Reporting Manual has been available since the early 1980s,[6] the terminology used

to describe respiratory care activities varies markedly among our hospitals.[7] Establishing systems to effortlessly collect data beyond what commonly exists in standard internal organizational financial reports generated by the finance department is the major hurdle. Even those fortunate enough to have good health care information systems often have difficulty getting the data they need because their organization has limited programming resources. Managers with respiratory care information systems that allow flexibility in their configuration have the fewest hurdles to overcome in gathering data. Establishing a comparative database and generating reports is relatively easy once decisions are made about performance indicators to be reported. The selection of appropriate efficiency and financial indicators that can help us demonstrate value of our services and the value of respiratory care practitioners in delivering those services is absolutely critical in our increasingly competitive health care environment. Although establishing performance indicators and a comparative database for respiratory care services may be a formidable task, we must get it done. Otherwise, it will become increasingly difficult to demonstrate the value of our services—the services that respiratory care professionals add to their organizations. Although many things impacting our profession today are outside of our control, this is something that we can control. During the past year, the AARC's Management Section has been and will continue to collect incidence data for ventilator-associated pneumonia and cost per service, from those willing to contribute. These data will be published in the Management Section Newsletter later this year. Although the AARC will continue this initiative and introduce others that will eventually provide the data that are needed, respiratory care practitioners need to make sure that they are well informed about the process of benchmarking. A wealth of papers, books, and seminars can provide a solid foundation in the key concepts of benchmarking. The January-February 1993 issue of *Healthcare Forum Journal** is an excellent place to begin.[8-13]

To paraphrase WC Fields, sometimes one must take the bull by the tail and face the situation. The article by Chatburn, El-Khatib, and Blumer in this issue is an example of just that. They gathered data on PICU ventilation, an area important to them, and are using those data as a benchmark to improve their clinical processes. They went one step further in reporting those results to the community of respiratory care professionals. Others who provide similar services now have data with which to compare their results. It is an excellent start in an area where much must be done.

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Safety and Efficacy of Oral Levofloxacin versus Cefuroxime Axetil in Acute Bacterial Exacerbation of Chronic Bronchitis

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OBJECTIVE: To compare the efficacy and tolerability of levofloxacin (the l-isomer of ofloxacin) 500 mg given orally once daily for 5-7 days with those of cefuroxime axetil 250 mg twice daily for 10 days. METHODS: Outpatients with a confirmed diagnosis of an acute bacterial exacerbation of chronic bronchitis (ABECB) were enrolled in this randomized, multicenter, open-label trial. Patients were evaluated before, during, and following therapy (5-7 days after termination of therapy). RESULTS: 248 patients received levofloxacin for a mean of 7 days and 244 received cefuroxime axetil for a mean of 10 days. Clinical response was rated a success (patient cured or improved) for 94.6% (210/222) of clinically evaluable levofloxacin-treated patients and 92.6% (212/229) of clinically evaluable cefuroxime axetil-treated patients. Microbiologic eradication rates by pathogen for microbiologically evaluable subjects were 97.4% for levofloxacin and 94.6% for cefuroxime axetil. Resistance to levofloxacin was found in 2% of isolates; 9% of isolates were resistant to cefuroxime. Both treatments were generally well tolerated with the most common adverse events in both groups affecting the gastrointestinal tract. CONCLUSIONS: When given orally, a once daily 500-mg dose of levofloxacin for 5-7 days was as effective and well tolerated as cefuroxime axetil (250 mg bid) given for 10 days to patients with ABECB. [Respir Care 1997;42(2):206-213]

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Additional investigators instrumental in the conduct of this multicenter trial are H Carveth, HM Faris, RA Fiddes, SM Garay, D Ginsberg, G Gomes, J Grossman, RN Hunt, B Interiano, WB Klaustermeyer, PE Korenblat, TW Littlejohn, M McAdoo, P McElvaine, NA Memon, RR Moyer, SV Nair, AD Puopolo, K Rice, RD Rosen, M Russell, WB Smith, JG Sullivan, WR Summer, A Thomas, JJ Upchurch, M Zervos, SK Zorn.

This study was presented, in part, at a satellite symposium of the International Congress of Chemotherapy in July 1995, Montreal, Quebec, and at the Infectious Disease Society of America in New Orleans, 1996.

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Introduction

Levofloxacin is the l-isomer of the racemic compound, ofloxacin. Levofloxacin is the microbiologically active isomer and demonstrates in vitro and clinical activity following either oral or intravenous (I.V.) administration. It is from two to four times more potent in vitro than isofloxacin, being active against most aerobic Gram-positive and Gram-negative organisms associated with respiratory infections. It is active against *Streptococcus pneumoniae*, *Staphylococcus aureus*, *Mycoplasma pneumoniae*, *Haemophilus influenzae*, *Chlamydia pneumoniae*, and *Moraxella catarrhalis*.[1-3] These in vitro findings suggest that levofloxacin should be active in the treatment of bacterial respiratory infections due to susceptible organisms.

Levofloxacin demonstrates almost 100% bioavailability following oral administration.[4] Steady-state peak plasma concentrations of 5.72 g/mL following once-daily 500 mg oral administration and 6.40 g/mL following once-daily 500 mg I.V. administrations are attained.[5] The drug is eliminated primarily through renal clearance, with up to 86% of an oral dose being found in the urine within 24 hours. Levofloxacin has

a plasma elimination half-life of approximately 7-8 hours. These pharmacokinetic characteristics support the administration of levofloxacin as a single once-daily intravenous or oral dose.

Levofloxacin penetrates well into bronchiolar tissue and sputum when administered to healthy subjects or to patients with lower respiratory tract infections.[6] The sputum concentration of levofloxacin following oral administration has been reported to range from approximately 85% to 95% of serum concentrations.[7,8] These findings suggest that clinically effective concentrations of levofloxacin are achieved at target respiratory sites, exceeding the minimum inhibitory concentrations (MICs) for common respiratory pathogens. Levofloxacin demonstrates broad-spectrum activity in vitro against respiratory pathogens. MIC90s for common pathogens are *Staphylococcus aureus* (methicillin-susceptible), 0.41 mg/L; *Streptococcus pneumoniae*, 1.91 mg/L; *H. influenzae*, 0.02 mg/L, and *M. catarrhalis*, 0.09 mg/L.[1]

Levofloxacin has a toxicity profile similar to its parent compound ofloxacin in animals; therefore, it is reasonable to expect good clinical activity without a change in the incidence of adverse events.

Preliminary published reports have found levofloxacin to be effective in the treatment of lower respiratory tract infections,[9] community-acquired pneumonia,[10,11] and acute bacterial sinusitis.[12] Based on these findings, a rigorous assessment of the clinical efficacy of levofloxacin in patients with acute bacterial exacerbations of chronic bronchitis (ABECB) was undertaken in this study.

Cefuroxime axetil is a widely used broad-spectrum β-lactam antibiotic effective against common pathogens associated with ABECB. Because of its wide spectrum of activity, it is often selected as primary empiric therapy for these infections.[13-15] The oral tablet and suspension, with a mean elimination half life of 1-2 hours, require twice-daily administration.

The objective of this randomized, active control, open-label multicenter study was to compare the efficacy and tolerability of levofloxacin 500 mg administered orally once daily for 5 to 7 days with those of cefuroxime axetil 250 mg administered orally twice daily for 10 days in adult outpatients with acute bacterial exacerbation of chronic bronchitis caused by susceptible organisms. Patients were selected based on guidelines developed by the Infectious Disease Society of America;[16] these criteria were used to design this study so that only patients with acute bacterial exacerbations of chronic bronchitis would be evaluated, rather than confounding the study by the inclusion of patients who might have been experiencing a viral bronchitis or a nonacute episode of bronchitis.

Patients & Methods

Patient Population

Male and female outpatients aged 18 years or older who were experiencing an episode of ABECB and who could receive oral medication were eligible for inclusion in the study. The diagnosis of ABECB was based on a history of chronic obstructive pulmonary disease (COPD), including chronic bronchitis and emphysema; a recent increase in cough; a change in the quality (eg, purulence and/or consistency) or increase in production of sputum; and physical findings consistent with ABECB. Patients who had received prior antibiotic therapy could be enrolled if prior therapy had lasted less than 24 hours or if the patient had not been stabilized or improved by prior therapy. Women were required to be postmenopausal, surgically sterile, or using an adequate form of birth control. Women with childbearing potential were required to have had a normal menstrual flow within 1 month before entering the study and a negative pregnancy test immediately before study entry.

Patients with a history of allergy or serious adverse reaction to levofloxacin, another quinolone, or any β-lactam antimicrobial were excluded from participation in the study. Other exclusion criteria included severe illness necessitating intravenous antibiotics or a second systemic antimicrobial; infection caused by an organism known at screening to be resistant to either study drug; and use of an investigational agent within the previous 30 days. In addition, potential patients with acute bronchitis, pneumonia, cystic fibrosis or any other disease interfering with evaluation of either study drug were excluded from study participation, as were potential patients with a creatinine clearance $\leq$ 50 mL/min, a seizure disorder, or an unstable psychiatric condition, or those who were pregnant or lactating.

Institutional review board approval was obtained and all patients gave written informed consent.

Study Design

This open-label study was conducted at 34 centers throughout the United States between August 1993 and May 1994. Patients were randomly assigned to receive either one 500-mg tablet of levofloxacin orally once daily for 5 to 7 days or one 250-mg tablet of cefuroxime axetil orally twice daily for 10 days. The duration of therapy could be extended at the discretion of the investigator with approval of the study sponsor. The use of other systemic antimicrobials during the study was prohibited. The use of other medications was kept to a minimum, particularly aluminum-based antacids and mineral supplements or vitamins with iron or minerals, which, if necessary, were administered at least 2 hours before or after levofloxacin because of possible decrease in bioavailability of levofloxacin associated with concomitant administration.

Upon entry into the study, patients provided a detailed medical history and underwent a physical examination, thoracic radiograph, and complete blood count (CBC), platelet count, serum chemistry, and urinalysis with microscopic examination of sediment. Gram's stain and culture were done on respiratory secretions collected within 48 hours before admission into the

study; susceptibility testing was performed on all bacterial isolates. Respiratory specimens included deep expectorated or suctioned sputum, transtracheal aspirates, or bronchial brushings, biopsies, or washings. If bacteremia was suspected, 2 samples for blood culture were submitted. Theophylline levels were monitored if indicated.

Patients returned for a scheduled visit and evaluation of clinical progress on Days 3-5 of study drug administration. Those patients with negative culture results on admission were allowed to continue in the study. Those with pathogens resistant to the study drug were allowed to remain in the study only if, in the opinion of the investigator, there had been reasonable clinical improvement during the study. Otherwise, the patient was discontinued from the study and considered to be a clinical failure. The post-therapy visit was scheduled for 5-7 days after termination of therapy at which time a physical examination and repeat laboratory testing were performed.

Efficacy Evaluations

Efficacy evaluations included assessment of clinical signs and symptoms, clinical response rates, and microbiologic eradication rates. The clinical response among patients evaluable for clinical efficacy was the primary efficacy parameter; microbiologic response in the group of patients evaluable for microbiologic efficacy was the secondary efficacy parameter.

The clinical signs and symptoms of bronchitis were assessed by the investigator at the time of admission into the study and at the post-therapy visit. Clinical symptoms of ABECB, including chills, chest pain, shortness of breath, increased cough, increased sputum, and purulent sputum, were graded as present or absent. Clinical signs of bronchitis on thoracic auscultation, including diminished breath sounds, rales, egophony, rhonchi, and wheezes, were graded as none, mild, moderate, or severe.

At the post-therapy visit the investigator evaluated the clinical response as cured, improved, failed, or unable to evaluate. A clinical cure was defined as resolution of the signs and symptoms associated with active infection; clinical improvement was defined as incomplete resolution of signs and symptoms with no further need for antimicrobial therapy. The clinical cure rate was defined as the percentage of clinically evaluable patients who were cured and the clinical success rates as the percentage improved. A lack of response to therapy was deemed a clinical failure.

If the patient could produce sputum, specimens were submitted for Gram's stain and culture and susceptibility testing at the post-therapy visit. The microbiologic response was assessed as eradicated, persisting, or unknown. Blood cultures were repeated post-therapy if bacteremia was found at admission. Patients were evaluable for clinical efficacy unless the course of therapy was too short, the diagnosis was unconfirmed, the patient was unevaluable for safety, an effective concomitant antibiotic was administered, a post-therapy culture or evaluation was not conducted at the appropriate time, or the patient violated other selected protocol criteria. Patients were considered to be microbiologically evaluable unless they met one of the following criteria: clinically unevaluable, the infection was not bacteriologically proven, or bacteriologic cultures were obtained at incorrect sampling intervals. The "course of therapy was too short" was defined as having received levofloxacin < 4 days or cefuroxime axetil for < 5 days. However, patients who failed therapy and completed more than 48 hours of therapy were considered evaluable for efficacy.

Susceptibility to levofloxacin and cefuroxime axetil was determined for all pathogens isolated at admission and at the post-therapy visit if cultures were obtained. Susceptibility testing was performed in accordance with the National Committee for Clinical Laboratory Standards methods. A broth microdilution susceptibility assay was used for determination of the minimum inhibitory concentration (MIC), which was the primary susceptibility criterion. The susceptibility ratings for levofloxacin were as follows: susceptible if MIC was ≤ 2.0 μg/mL, moderately susceptible if MIC 4.0 μg/mL, and resistant if MIC ≥ 8.0 μg/mL. Organisms were classified as susceptible to levofloxacin if the inhibitory zone diameter was ≥ 16 mm, as moderately susceptible if the diameter was 13-15 mm, and as resistant if the diameter was ≤ 12 mm.

Safety Evaluations

Safety evaluations included assessment of the incidence of treatment-emergent adverse events; results of laboratory testing, including hematology, serum biochemistry, and urinalysis; and physical examination findings, including vital sign measurements. The severity of adverse events was graded as mild, moderate, or severe, and the relation of adverse events to treatment was assessed by the investigator as none, remote, possible, probable, or definite. Adverse events were classified as serious if they presented a significant threat to the well-being of the patient. These included any event that was fatal, life-threatening, permanently or significantly disabling, required hospitalization or prolonged hospitalization, resulted in long-term outpatient treatment, or resulted in a congenital anomaly, cancer, or overdose.

Statistical Methods

Comparison of continuous demographic variables was performed using a 2-sided t test for independent samples, and comparison of categorical variables was performed using a 2-sided Fisher's exact test. In addition to clinical response among clinically evaluable patients and microbiologic response among microbiologically evaluable patients, the efficacy analyses were performed for the intent-to-treat subjects (all subjects assigned to treatment groups as randomized) and the modified intent-to-treat subjects (all subjects assigned to treatment groups based on actual treatment received). A 2-sided 95%

confidence interval (CI) was calculated around the difference in clinical success rates (percentage of patients cured or improved) for the 2 study drugs. Results were compared for each study center enrolling 10 or more subjects per treatment group. Two-sided 95% CIs were also computed around the differences in overall microbiologic eradication rates by pathogen, pathogen category, and study centers that enrolled 10 or more evaluable patients in each treatment group. For both the clinical success rates and microbiologic eradication rates, results were pooled from study centers that enrolled fewer than 10 evaluable patients in each treatment group.

All patients with postadmission data were included in the safety analyses. Ninety-five percent CIs were calculated around the between-treatment difference in patient incidence rates and adverse event rates for each body system. A Kolmogrov-Smirnov test was used to compare the 2 treatment groups with respect to cumulative distribution of percentage changes in laboratory test results from admission to post-therapy.

Table 1. Demographic and Baseline Characteristics of the Modified Intent-To-Treat* Patient Population

Subject Characteristics	Levofloxacin (n = 248)	Cefuroxime axetil (n = 244)	Total (n = 492)
Gender			
Male	124 (50%)	140 (57.4%)	264 (53.7%)
Female	124 (50%)	104 (42.6%)	228 (46.3%)
Race			
Caucasian	181 (73%)	177 (72.5%)	358 (72.8%)
Black	40 (16.1%)	45 (18.4%)	85 (17.3%)
Hispanic	26 (10.5%)	20 (8.2%)	46 (9.3%)
Asian	1 (0.4%)	0	1 (0.2%)
Other	0	2 (0.8%)	2 (0.4%)
Age (years)			
Mean (SD)	51.7 (18.0)	53.1 (17.5)	52.4 (17.8)
Range	18-97	18-87	18-97
COPD	221 (89.1%)	219 (89.8%)	440 (89.4%)

*The modified intent-to-treat approach groups patients according to treatment actually received. COPD = chronic obstructive pulmonary disease.

Results

Patient Disposition

Total enrollment at 34 centers included 492 patients, of whom 248 received levofloxacin and 244 received cefuroxime axetil. Demographic and baseline characteristics were comparable for the treatment groups (Table 1). Patient disposition is summarized in Table 2. The mean duration of therapy was 7 days for levofloxacin-treated patients and 10 days for cefuroxime axetil-treated patients. The groups were comparable with respect to the types of concomitant therapies used.

Efficacy

Clinical Response. The clinical response was rated a success (patient cured or improved) for 94.6% (210/222) of clinically evaluable patients who received levofloxacin and for 92.6% (212/229) of those who received cefuroxime axetil (Fig. 1). The individual confidence intervals for all of the analysis groups were centered below zero and are consistent with therapeutic equivalence of the treatments regarding clinical success rates.

With the exception of shortness of breath, which resolved in 69.7% of levofloxacin-treated and 72.4% of cefuroxime axetil-treated patients, all of the symptoms of bronchitis were resolved in more than 86% of patients reporting them at study admission. Abnormalities on thoracic auscultation were resolved or improved in 85% or more of patients demonstrating them at admission, with the exception of diminished breath sounds, which were resolved or improved in 62.5% of levofloxacin-treated patients and 55.2% of cefuroxime axetil-treated patients.

Table 2. Disposition of Patients Entered into Antibiotic Comparison Study

Outcome	Levofloxacin	Cefuroxime	Total
	Number of Patients (%)		
Randomized	246	246	492
Drug actually dispensed	248	244	492
Completed therapy	230 (92.7)	225 (92.2)	455 (92.5)
Clinically evaluable	222 (89.5)	229 (92.2)	455 (91.7)
Microbiologically evaluable	134 (54.0%)	147 (60.2)	281 (57.1)
Evaluable for safety	243(98.9)	241 (98.8)	484 (98.4)
Basis for premature discontinuation			
Adverse event	7	8	15
Clinical failure	0	4	4
Protocol violation	2	1	3
Lost to follow-up	9	6	15
Total prematurely discontinued	18 (7.3)	19 (7.8)	37 (7.5)

*Percentage based on drug actually received.

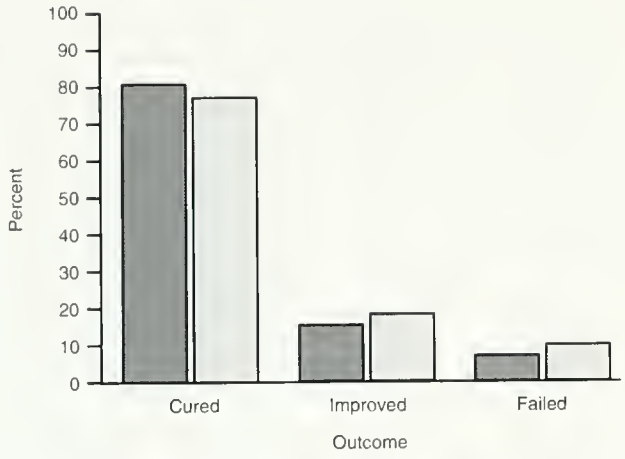

Fig. 1. Response rates (%) for clinically evaluable patients treated with Levofloxacin ( ) and Cefuroxime ( ).

Microbiologic Response. Pathogens were isolated at admission from 145 of the levofloxacin and 156 of the cefuroxime axetil groups. Table 3 shows the most common pathogens and the clinical success rates.

The microbiologic eradication rates (for microbiologically evaluable patients) by pathogen were 97.4% in the levofloxacin treatment group and 94.6% in the cefuroxime axetil treatment group, while the eradication rates by patient were 96.3% and 93.2%, respectively. Microbiologic persistence was observed in 5 (3.7%) levofloxacin-treated patients and 10 (6.8%) cefuroxime axetil-treated patients. Overall, the most prevalent pathogens in both treatment groups were Gram-negative aerobes (77.4% of all pathogens in levofloxacin-treated patients and 72.1% in cefuroxime axetil-treated patients); the remainder were Gram-positive aerobes. Microbiologic eradication rates by pathogen category and pathogen are summarized in Table 4.

Table 3. Clinical Response Rates for Patients with Pathogens of Primary Interest (Clinically Evaluable Patients)

Pathogen	Levofloxacin			Cefuroxime axetil		
	n*	Cured/Improved n (%)	Failed n (%)	n*	Cured/Improved n (%)	Failed n (%)
Haemophilus influenzae	44	42 (95)	2 (5)	31	31 (100)	0
Haemophilus parainfluenzae	27	26 (96)	1 (4)	32	29 (91)	3 (9)
Moraxella (Branhamella) catarrhalis	25	24 (96)	1 (4)	32	28 (88)	4 (13)
Streptococcus pneumoniae	16	14 (88)	2 (13)	10	10 (100)	0
Staphylococcus aureus	10	9 (90)	1 (10)	35	33 (94)	2 (6)

*Number of patients from whom pathogen was isolated either singly or in combination.

Table 4. Microbiologic Eradication Rates Summarized by Pathogen Category and Pathogen for Microbiologically Evaluable Patients

	Levofloxacin		Cefuroxime axetil		95% CI*
	n	Eradicated	n	Eradicated	
Pathogen Category					
Gram-positive aerobe	43	41 (95%)	62	60 (97%)	[-7.4, 10.3]
Gram-negative aerobe	147	144 (98%)	160	150 (94%)	[-8.9, 0.5]
Total by pathogen	190	185 (97%)	222	210 (95%)	[-6.8, 1.2]
Total by patient	134	129 (96%)	147	137 (93%)	[-8.6, 2.5]
Pathogen					
Haemophilus influenzae	44	42 (96%)	31	29 (94%)	
Haemophilus parainfluenzae	27	27 (100%)	32	30 (94%)	
Moraxella catarrhalis	25	25 (100%)	32	29 (91%)	
Streptococcus pneumoniae	16	14 (88%)	10	10 (100%)	
Staphylococcus aureus	10	10 (100%)	35	34 (97%)	
Pseudomonas aeruginosa	10	9 (90%)	9	8 (89%)	
Escherichia coli	8	8 (100%)	6	6 (100%)	
Streptococcus Group C	5	5 (100%)	5	4 (80%)	
Streptococcus milleri	4	4 (100%)	5	5 (100%)	
Klebsiella pneumoniae	3	3 (100%)	11	10 (91%)	
Haemophilus parahemolyticus	2	2 (100%)	5	4 (80%)	
Serratia marcescens	1	1 (100%)	5	5 (100%)	
Neisseria meningitidis	0	0	5	5 (100%)	

*Two-sided confidence interval around the difference (cefuroxime axetil minus levofloxacin) in microbiologic eradication rates.

The pattern of in vitro susceptibility of pathogens isolated at admission was somewhat different between the two treatment groups. Of 202 pathogens with known in vitro levofloxacin susceptibility isolated from levofloxacin-treated patients, 197 (98%) were susceptible in vitro, 1 (<1%) was moderately susceptible, and 4 (2%) were resistant. The corresponding in vitro cefuroxime axetil susceptibilities for 227 isolates from cefuroxime axetil-treated patients were 191 (84%), 15 (7%), and 21 (9%).

Three patients in the levofloxacin group and 4 patients in the cefuroxime axetil group developed superinfections (8 pathogens). For these 8 isolates, 6 were susceptible to levofloxacin (2 unknown susceptibility) and 5 were susceptible to cefuroxime axetil (1 moderately susceptible and 2 of unknown susceptibility).

The clinical response and microbiologic eradication rates were comparable across the relevant analysis groups of clinically evaluable, microbiologically evaluable, and modified intent-to-treat. Moreover, the results were consistent across study centers. Among microbiologically evaluable patients in whom the pathogen was eradicated, clinical cure or improvement was observed for 126 of 129 (97.7%) levofloxacin-treated patients and in 135 of 137 (98.5) cefuroxime axetil-treated patients, indicating excellent concordance between clinical and microbiologic results.

Safety

The incidence of treatment-emergent adverse events was comparable for the treatment groups (Table 5). No deaths occurred during the study.

Fifteen patients (7 levofloxacin-treated and 8 cefuroxime axetil-treated) discontinued the study because of an adverse event. Nine levofloxacin-treated patients and five cefuroxime-treated patients reported serious or potentially serious adverse events, all of which were deemed to be unrelated to the study drug by the investigators.

There were no clinically significant treatment-emergent mean changes from admission to post-therapy for any laboratory test result in either treatment group. The incidence of markedly abnormal test results for individual analyses within a given treatment group was low (≤ 2.2%) and comparable between treatment groups. Moreover, there were no clinically significant treatment-emergent physical examination abnormalities or changes in vital signs that were considered to be related to study drug administration.

Discussion

The results of this comparative study indicate that a once-daily 500-mg dose of levofloxacin given for 5-7 days is as effective as cefuroxime axetil, 250 mg twice daily, given orally for 10 days for the treatment of acute exacerbations of chronic bronchitis.

In actual clinical practice, physicians typically treat ABECB empirically, before the results of bacterial cultures and susceptibility testing are available. The choice of an appropriate antibiotic is determined by a variety of factors including its spectrum of activity, the pathogens most likely to cause the infection, the local pattern of bacterial resistance, and the overall empiric effectiveness of the antibiotic against pathogens associated with lower respiratory tract infections. The growing incidence of β-lactam and/or macrolide resistance in common bacterial pathogens such as *Streptococcus pneumoniae*, *Staphylococcus aureus*, and *H. influenzae* further complicates the selection of an appropriate antibiotic for initial empiric therapy.

In this study, the most common pathogens were *H. influenzae*, *H. parainfluenzae*, *M. catarrhalis*, *Streptococcus pneumoniae*, and *Staphylococcus aureus*. These are among the pathogens most commonly associated with acute bacterial exacerbations in patients with chronic bronchitis[3] and are consistent with the organisms clinicians could expect to encounter routinely. Traditional antibiotics are exhibiting diminishing clinical and microbiologic activity against these common respiratory pathogens. For example, in one survey of respiratory isolates obtained from patients in the United States, 17% of *H. influenzae* and 84% of *M. catarrhalis* were resistant to penicillin, ampicillin, and amoxicillin.[17] Reports from Canada indicate that approximately 8% of isolates of

Table 5. Incidence of Drug-Related Adverse Events* Reported by 1% or More of Patients Evaluable for Safety

Adverse Event	Levofloxacin (n = 243)				Cefuroxime axetil (n = 241)			
	Mild	Moderate	Severe	Total	Mild	Moderate	Severe	Total
Vaginitis†	2	3	0	5 (4)‡	0	2	0	2 (2)
Nausea	5	0	1	6 (3)	2	0	0	2 (1)
Diarrhea	4	0	0	4 (2)	2	3	1	6 (3)
Taste Perversion	2	0	0	2 (1)	4	0	0	4 (2)
Total	16	7	2	24 (10)	11	8	2	19 (8)

*Adverse events considered by the investigator to be probably or possibly related to study drug administration.
†Percentage calculated from the total number of women evaluable for safety in each group (levofloxacin women = 122, cefuroxime axetil women = 102).
‡Numbers in parentheses = percentages of evaluable patients experiencing adverse events

Streptococcus pneumoniae in one series were penicillin-resistant; penicillin-susceptible strains were generally susceptible to other antimicrobial agents, but a high proportion of penicillin-resistant isolates were also resistant to tetracycline (63%), erythromycin (50%), and cefuroxime (38%).[18] In a recent survey of 491 bacteremic isolates of *Streptococcus pneumoniae*, penicillin resistance was noted in 7.7% of isolates (5/491 with high-level resistance and 33/491 with intermediate resistance; only a small proportion of the isolates were resistant to fluoroquinolones). Of the 491 isolates, 453 were susceptible to penicillin, 469 were susceptible to ofloxacin, and 452 were susceptible to ciprofloxacin; intermediate resistance was noted for 33 isolates to penicillin, 16 to ofloxacin (the parent compound of levofloxacin) and 33 to ciprofloxacin; high-level resistance was noted for 5 isolates to penicillin and ofloxacin and 6 to ciprofloxacin (Data on file, The RW Johnson Pharmaceutical Research Institute). A recent analysis of respiratory pathogens isolated from patients with pneumonia indicated that the majority of pathogens (104/182, 57%) had MICs of 0.250 μg/mL or lower for levofloxacin (Data on file, The RW Johnson Pharmaceutical Research Institute). Such findings indicate that common pathogens associated with ABECB may have a high probability of demonstrating resistance to traditional antibiotic regimens[19] and will be susceptible to levofloxacin.

In this context, it is noteworthy that of the 437 pathogens isolated on admission in our study, only 8 were resistant to levofloxacin compared to 33 that were resistant to cefuroxime axetil. Of the 33 pathogens resistant to cefuroxime axetil, 27 were completely susceptible to levofloxacin. This broad spectrum of activity suggests that levofloxacin can provide appropriate coverage of respiratory pathogens, including *Streptococcus pneumoniae*, when used as empiric therapy.

The previous clinical experience with levofloxacin in the treatment of ABECB indicates that it is highly effective.[20,21] Results from a recent trial comparing oral levofloxacin 500 mg once daily to cefaclor 250 mg three times daily in patients with ABECB found that among clinically evaluable patients treated with levofloxacin, 72.1% were cured and 19.5% improved compared with 64.5% cured and 27.1% improved with cefaclor. The overall microbiologic eradication rates by pathogen reveal a slightly higher eradication rate for levofloxacin (95%) compared to cefaclor (86.5%). There was 100% eradication of *H. influenzae*, 94.7% eradication of *M. catarrhalis*, and 90% eradication of *Streptococcus pneumoniae* in the levofloxacin group compared to eradication rates of 70.8%, 100%, and 85.7%, respectively, in the cefaclor group. These findings indicate that levofloxacin given for 5-7 days was as effective as cefaclor given for 7-10 days for ABECB.[9] The findings for levofloxacin were similar to those in our study, with excellent activity against a broad range of respiratory pathogens.

Cefuroxime axetil has been shown to be effective in the treatment of lower respiratory infections.[13,15,22] When administered as a 250-mg twice-daily dose to patients with acute bronchitis or ABECB, clinical cure or improvement was observed in 109 of 143 (76%) patients.[15] Among hospitalized patients with lower respiratory tract infections, initial treatment with I.V. cefuroxime followed by oral cefuroxime axetil resulted in cure or improvement in 223 of 256 (87%) patients.[22] In our study, the overall clinical response rate was 94.6% (210/222) for levofloxacin-treated patients and 92.6% (212/229) for those receiving cefuroxime axetil. However, a recent report[18] that 38% of penicillin-resistant strains of *Streptococcus pneumoniae* were also cefuroxime resistant (MIC $\geq$ 2 g/mL) suggests that resistance to this drug and other β-lactams is increasing, posing the threat of a future loss of activity of these drugs against this pathogen. β-lactams should therefore be used judiciously for the empiric treatment of ABECB, particularly in regions in which drug resistance is increasing.

Although it has been recognized that levofloxacin demonstrated good in vitro activity against respiratory pathogens,[1] including the so-called atypical pathogens that account for a growing proportion of cases of ABECB, our study confirms the broad spectrum of activity of this antibiotic in patients with ABECB.

In our study, the mean duration of treatment was 7 days in the levofloxacin group compared to 10 days in the cefuroxime axetil group. This finding suggests that a significant clinical response can occur with a shorter treatment regimen of levofloxacin than cefuroxime axetil. Although the duration of treatment of ABECB is often determined by the physician based on the patient's clinical status, these findings indicate that many patients will derive full benefits from a 5-7 day course of levofloxacin therapy. Together with the ease of once-daily administration, this may enhance patient compliance beyond that observed with traditional empiric antibiotic therapies.

The safety profiles of levofloxacin and cefuroxime axetil were comparable, and both drugs were generally well tolerated. In controlled Phase II/III clinical trials in more than 2,400 patients treated with levofloxacin, the antibiotic was well tolerated with 5.6% of patients reporting a drug-related adverse event. The only drug-related events that occurred in >1% of levofloxacin-treated patients were diarrhea (1.1%) and nausea (1.4%).

Conclusions

The results of this study indicate that oral levofloxacin is as effective and well tolerated as cefuroxime axetil for the treatment of patients with ABECB. Furthermore, because of the increasing prevalence of common respiratory pathogens that are demonstrating resistance to traditional antibiotics, treatment with penicillins, β-lactams, erythromycins, and tetracyclines may not provide the broad-spectrum coverage required from empiric treatment. In this regard, empiric treatment with a broad-spectrum fluoroquinolone may offer wider coverage of common pathogens. Levofloxacin was well tolerated in this

study and was associated with a lower incidence of diarrhea than cefuroxime axetil. The short course of once-daily levofloxacin treatment provided convenient, effective and well-tolerated treatment for ABECB.

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Acute Respiratory Failure from Neck Hematoma Following Venous Cannulation in a Patient Receiving Streptokinase

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I present a case of acute respiratory failure, caused by hematoma in the neck that led to compression of the upper airway and possible cerebral ischemia. A few hours after the patient with acute myocardial infarction had been treated with intravenous systemic infusion of streptokinase, heparin, and oral acetylsalicylic acid, several attempts were made to gain intravenous access by percutaneous puncture of the internal jugular vein, guided by external anatomic landmarks. A large hematoma developed in the neck, causing acute respiratory failure. The patient's condition was detected during close monitoring, and the patient was treated in time by orotracheal intubation and mechanical ventilation. Intravenous heparin was discontinued, and red blood cells and fresh frozen plasma were transfused. The main goal of thrombolytic therapy—reperfusion with early peak of creatine kinase was not achieved. The case suggests that in the first few hours after treatment of acute myocardial infarction with systemic intravenous thrombolytic therapy, the hemostatic defect that develops increases the risk for bleeding after percutaneous puncture of internal jugular vein. The procedure is safer when it is performed with caution by the most skillful and experienced operator and after prior ultrasonic examination of the neck anatomy in order to avoid several puncture attempts and the increased likelihood of abundant hemorrhage at the puncture sites. Close monitoring is essential to identify and treat any potential life-threatening complication. [Respir Care 1997;42(2):214-217]

Introduction

Percutaneous cannulation of the internal jugular vein guided by external anatomic landmarks is the preferred access to this central vein in emergency situations. It has many advantages, and it can be performed during cardiopulmonary resuscitation. The control of bleeding is supposed to be easier even in bleeding diatheses.[1-3] It can be performed rapidly (with an access time averaging only 44.5 seconds (range 1-1,000 seconds) from the skin surface to the vein. The success rate is up to 88%. On average, 3 attempts are necessary to reach the vein successfully (range 1-28 attempts). A complication rate of 5-10% is expected with this method. The incidence of carotid artery puncture is up to 8.3%, of hematoma up to 3.3%, of brachial plexus irritation or injury up to 1.7%, and of pneumothorax up to 1.7%.[4] In experienced hands, this method is safe even in bleeding diatheses.[5,6] A rare complication (< 0.1%) is an abundant neck hematoma at the puncture site, resulting in severe compression of the upper airway with respiratory arrest and altered mental status.[1]

Occlusive coronary thrombi are present in 90% of acute myocardial infarctions. Such thrombi can be dissolved by fibrinolytic agents, especially in the first few hours following the infarction. The result is recanalization of the previously occluded coronary artery. In 30-70% of cases, reperfusion of jeopardized myocardium is achieved, thus limiting infarct size,

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improving postinfarction left-ventricular function, and decreasing the incidence of sudden cardiac death and extension of ischemic necrosis.[7-11]

Streptokinase is the most widely used thrombolytic agent. It is effective, easy to administer intravenously, and inexpensive. Other advantages of streptokinase are (1) the lowest incidence of disabling hemorrhagic stroke, and (2) effective treatment up to 12 hours after symptoms of acute myocardial infarction appear and in cardiogenic shock.[10,11] Streptokinase is a protease produced by β-hemolytic streptococci, with a half life of 23 minutes.[7]

Criteria for streptokinase therapy of patients with acute myocardial infarction are fulfilled when precordial chest pain lasting more than 30 minutes, but less than 12 hours, has not responded to 2 to 3 sublingual nitroglycerin tablets and when, at least, 1-2 mm of ST-segment elevation is present in at least 2 leads of the electrocardiogram. Excluded are patients with contraindications (recent severe trauma, a major surgery, or organ biopsy, arterial puncture or intramuscular injection within a few hours, terminal stage of a chronic or malignant disease, gastrointestinal ulcer, or active internal bleeding, a known bleeding diathesis, stroke in last 6 months, untreated and unbalanced arterial hypertension with systolic blood pressure over 220 mm Hg and diastolic blood pressure over 120 mm Hg, current anticoagulant therapy, any condition in which bleeding is a considerable hazard or would be difficult to manage because of its location, allergy to streptokinase, heparin, or acetylsalicylic acid, or streptococcal infection within recent months.[7,8,10]

The recommended dose of streptokinase is 1,500,000 IU, given intravenously within 1 hour. Streptokinase activates plasminogen to plasmin indirectly, forming streptokinase-plasminogen complex, which is capable of activating the remaining circulating and thrombus-bound plasminogen to plasmin. Plasmin degrades primary components of vascular thrombi fibrin and also circulating fibrinogen, Factor V and VIII.[7,8,12]

To prevent reinfarction, systemic intravenous infusion of heparin for 24 to 48 hours and oral acetylsalicylic acid are recommended. Most short-term infusions of streptokinase do not require laboratory monitoring.[8,13,14]

On the other hand, effective clot lysis with reperfusion can lead to catastrophic bleeding because protective thrombi are dissolved as well. The risk for bleeding is increased up to 5%, after early invasive procedures—percutaneous cannulation of central veins included—even up to 15%.[13,14]

I report a case of acute respiratory failure and subsequent coma associated with streptokinase administration and central venous catheter placement.

Case Summary

A 65-year-old woman, who previously had suffered an anterolateral myocardial infarction, was admitted to the hospital because of precordial chest pain that had lasted about 90 minutes and had not responded to 2-3 sublingual nitroglycerin tablets. At admission she was dyspneic, her blood pressure was 90/60 mm Hg, and her pulse rate was 70/min. The electrocardiogram (ECG) recorded a sinus rhythm, horizontal axis, rate 67/minute; 4-mm ST-segment elevations in leads II, III, and aVF; and 1-2-mm ST-segment elevations in the leads V2 and V3. QRS complexes were widened (0.12 s). Admission red blood cell count was $3.64 \cdot 10^{12}$/L, hemoglobin 121 g/L (ie, 12.1 g/dL), platelets $144 \cdot 10^{9}$/L. Creatine kinase, lactic dehydrogenase, and prothrombin time were normal.

Acute inferoposterior myocardial infarction was suspected. The patient was admitted to the ICU for continuous ECG monitoring and treatment. A peripheral intravenous line was inserted. Because there were no contraindications, the patient was treated with high dose (1,500,000 IU), short-term (60 min) systemic intravenous infusion of streptokinase, 100 mg oral acetylsalicylic acid, and heparin (5,000 IU intravenous bolus before the start of streptokinase infusion, followed by intravenous infusion of 1,000 IU/h for the next 24 h).

Ventricular arrhythmias of Lown's class II, III, IVB developed 5 hours later. They disappeared after administration of 2 mg/min of intravenous 2% lidocaine. Soon thereafter, atrial flutter with ventricular rate 40/min and widened QRS complexes (0.13 s) were observed. The patient became dyspneic with blood pressure of 110/70 mm Hg. Pulse rate increased to 50/min after 0.5 mg intravenous atropine. By this time, the intravenous line was no longer patent. All attempts to gain another peripheral intravenous access failed. An anterior-approach, sterile percutaneous cannulation of the internal jugular vein guided by external anatomic landmarks was performed. After several unsuccessful attempts, the central intravenous catheter was inserted via the guide wire by the Seldinger method. Immediate chest fluoroscopy confirmed the location of the tip of the catheter in the superior cava.

Three hours later swelling was noticed at the puncture site of the neck. One hour later, it had spread to the upper part of the chest and lower jaw. Hematoma was suspected and intravenous heparin was stopped. Within 2 hours, dyspnea, cyanosis, hypoventilation, inspiratory stridor, and coma (Glasgow coma scale 7-9) had developed and were thought to be the consequence of abundant neck hematoma at the puncture site. Arterial blood was not analyzed because it was believed that puncture of a peripheral artery would lead to further bleeding. Although an orotracheal tube was inserted, the patient's mental status did not improve and hypoventilation persisted. Mechanical ventilation had to be started (intermittent positive pressure ventilation, with F_{IO_2} 0.40, ventilatory rate 12-15/min). At that time, control red blood cell count was $2.88 \cdot 10^{12}$/L, hemoglobin 93 g/L, hematocrit 0.277. The patient was given 350 mL of packed red blood cells and 200 mL of fresh frozen plasma.

During the next 2 hours, the Glasgow coma scale gradually increased to 10, but it was 24 hours before the patient was fully oriented and breathing spontaneously although still intu-

bated. The presence of a cuff leak 36 hours after the start of thrombolytic therapy demonstrated that the neck hematoma had been resorbed to the extent that orotracheal tube could be removed. At that time, control red blood cell count was $3.09 \cdot 10^{12}$/L, hemoglobin 103 g/L, hematocrit 0.291, and platelets $98 \cdot 10^{9}$/L. Although still dyspneic during the next few hours, the patient was fully oriented and suffered no neurologic sequelae. Blood pressure was 100/60 mm Hg and pulse rate 60-70/min. On chest fluoroscopy, venous congestion and an enlarged left atrium and ventricle were seen. The ECG demonstrated atrial fibroflutter with widened QRS complexes (0.12 s), ventricular rate 67/min, and signs of inferoposterior and anterolateral myocardial infarction (Q wave in II, III, and aVF; absent R wave in V1-V4; and T-wave changes in V5-V6).

Streptokinase treatment was deemed unsuccessful because peak creatine kinase value was reached 18 hours after the start of thrombolytic therapy, and no ventricular arrhythmias were recorded during streptokinase infusion. In spite of unsuccessful fibrinolytic therapy and life-threatening hemorrhage with acute respiratory failure, the patient survived and was discharged from the hospital 5 weeks later in class III according to the NYHA classification (ie, New York Heart Association).

Discussion

After short-term (60 min) systemic intravenous infusion of 1,500,000 IU of streptokinase is completed, all of the circulating plasminogen, 87% of administered streptokinase, and all native fibrinogen disappear. α-2-antiplasmin is decreased to 20%, and products of fibrinogen and fibrin degradation are increased 14-fold. Because streptokinase-induced plasminogen-activator activity is present in circulation for several hours once the infusion of streptokinase is terminated, the reappearance of circulating coagulation proteins is delayed. After 4 hours, only trace amounts of activators are present, native fibrinogen is reappearing in plasma, and normal fibrin in the clot and thrombin-coagulable proteins are increased. Therefore, the lytic effect still persist for 4 hours before the recovery starts. Considerable recovery is achieved 25 hours after the start of streptokinase therapy.[12]

The addition of intravenous heparin for the next 24 to 48 hours is sufficient to prevent rethrombosis without causing bleeding in a majority of cases, but prolonged activated-partial-thromboplastin time and thrombin time increase the risk for hemorrhage. Up to 24 hours, but especially in the first 4 hours after streptokinase therapy, the risk for bleeding is increased up to 15% with interventional techniques including percutaneous puncture of central veins. The protective hemostatic clot is not formed at the site of the injured vessel and bleeding in surrounding, extravascular tissues is the consequence.[12-14]

In our patient, the internal jugular vein was cannulated 5 hours after the start of streptokinase therapy when the hemostatic defect was pronounced. With the anterior approach, percutaneous puncture of the internal jugular vein is performed at the apex of the triangle between the two heads of the sternocleidomastoid muscle and the right clavicle. The internal carotid artery and upper respiratory airway (hypopharynx, larynx, and cervical trachea) lie medially.[15,16]

With several passages of the needle from the skin to the internal jugular vein, numerous smaller vessels of the area and the internal carotid artery were eroded, predisposing all to active hemorrhage in the first few hours after streptokinase therapy. Profound swelling of the neck, lower jaw, and upper thorax suggested the accumulation of extravascular blood. Inspiratory stridor, dyspnea, hypoventilation, cyanosis, and obtundation indicated compression of the upper airway, most probably of the hypopharynx, which consists of soft tissue.[16] Compression of the cervical trachea was less probable because of the trachea's anterior cartilaginous rings with the more pressure-vulnerable membranous portion located posteriorly. Impairment of venous and lymphatic drainage of the upper airway by hematoma could contribute to upper airway compression.[17]

The change in sensorium was attributed to upper airway obstruction, but some clinical evidence for cerebral ischemia (namely, failure of the patient's mental status to improve and hypoventilation to be corrected), which may have been due to partial occlusion of the right or even both internal carotid arteries, was present. The patient had to be mechanically ventilated for the next 24 hours.

Elective catheterization of the internal jugular vein is highly successful, but catheterization in emergency situations is less so. The complication rate for percutaneous cannulation of the internal jugular vein guided by external anatomic landmarks depends on the experience and ability of the operator and on anatomic variations in the position of the internal jugular vein. As with any interventional technique, more attempts and longer times are required in inexperienced and inept hands to gain access to the internal jugular vein. With more attempts and longer access times to the central vein the complication rate—puncture of the carotid artery, hematoma, or pneumothorax formation—is increased as well.[3-6,18] Therefore, many authors recommend the use of ultrasound and Doppler localization of the vein in special circumstances.[4,18] Acquired and temporary hemostatic defect after streptokinase therapy for acute myocardial infarction may be such a special circumstance.

Hematoma in this patient's neck was a serious side effect of treatment, resulting in acute respiratory failure that could be managed only by orotracheal intubation and mechanical ventilation. It jeopardized the patient, who had already suffered acute myocardial infarction. Close monitoring was of extreme importance in order to treat acute respiratory failure in time.

Active bleeding at the puncture site stopped a few hours after heparin had been discontinued and fresh frozen plasma given. The estimated blood loss of our female patient who weighed 60 kg was about 1,000 mL, according to admission

values of hemoglobin and control hemoglobin values several hours after the start of bleeding and 24 hours later. In anemia due to hemorrhage, a hemoglobin level of 100 g/L and hematocrit of 0.30 are enough to support aerobic metabolism in otherwise healthy subjects.[19] However, in patients with diseased coronary or cerebral arteries, any decrease in oxygen delivery may be damaging. They may require earlier transfusions than other patients, especially if they have high-grade coronary stenosis.[20] For these reasons, clinical judgment of our patient's status was more important than absolute hemoglobin value in the decision to transfuse 350 mL of packed red blood cells.

About 36 hours after the start of therapy, the patient could easily be extubated because the hematoma was resorbing and no further bleeding was expected, based on the pharmacokinetics of intravenously administered streptokinase and on the grounds of clinical judgment, including cuff-leak test.[21]

Reperfusion, which is the aim of thrombolytic treatment, was not achieved because an early peak of creatine kinase (within 12 hours of therapy), the mark of successful therapy, was not present.[22] The unsuccessful thrombolytic therapy could have been the consequence of discontinuing heparin and transfusing red blood cells and plasma.

Because there is an increased bleeding tendency up to 24 hours after the start of systemic intravenous thrombolytic therapy in acute myocardial infarction, this case suggests that when access to the internal jugular vein is needed during this time, it must be performed cautiously after prior ultrasonic localization of the internal jugular vein to make repeated attempts unnecessary and to avoid carotid artery puncture, hematoma formation at the puncture site, or other potentially catastrophic complications. Close monitoring of the patient is essential after the central jugular catheter is inserted, to detect complications and allow treatment before complications become life threatening.

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CO_2 Retention Secondary to Laparoscopic Cholecystectomy

David F Mayo RRT, Edwin C Kohl RRT, and Charles G Durbin Jr MD

Carbon dioxide (CO_2) is insufflated into the peritoneal cavity to facilitate laparoscopic surgery because it does not support combustion and, thus, allows safe use of electrocautery. Any residual pneumoperitoneum at the end of the procedure is rapidly absorbed due to the high solubility of CO_2 in blood. Massive amounts of CO_2 are absorbed occasionally and can precipitate hypercarbic respiratory failure. We report a case of severe hypercarbia associated with subcutaneous insufflation of CO_2 during laparoscopic cholecystectomy. Although the patient had cardiovascular disease, she experienced no serious consequences but did require postoperative mechanical ventilation. [Respir Care 1997;42 (2):218-220]

Introduction

The first laparoscopic cholecystectomy was performed in France by Dubois in 1987.[1,2] Today, laparoscopic surgery is being enthusiastically embraced as a "minimally invasive" alternative to open laparotomy. Its small incisions and reduced operative dissection have made postoperative convalescence swifter and less complicated.[3,4] Initially believed to be a safer alternative to an open procedure, numerous complications have now been reported.[5-9]

Laparoscopic cholecystectomy is performed with the aid of induced pneumoperitoneum. In the operating room, the surgeon makes an incision through the umbilicus and places and secures a hollow trocar through it. The patient's abdomen is insufflated with gas to allow visualization, dissection, and removal of the gallbladder guided by a fiberoptic endoscope. Because electrocautery is used to cut tissues and coagulate blood vessels during the procedure, carbon dioxide (CO_2) is the gas used to establish and maintain the pneumoperitoneum because it does not support combustion.[10] Although helium would meet these requirements, CO_2 readily dissolves in plasma, diminishing the consequences of inadvertent vascular gas embolism.[11,12] Adverse hemodynamic effects associated with abdominal distention are expected and have been well described.

Approximately 10 L of CO_2 is introduced initially to establish the pneumoperitoneum, and then a pressure-controlled servo mechanism supplies gas flow to maintain distention and keep intraperitoneal pressure about 15 cm H_2O. During the procedure as much as 100 L or more of CO_2 gas may be used. Much of this is lost to the atmosphere by system leaks or during surgical instrument placement through the hollow trocars. However, because CO_2 is absorbed through the peritoneal surface, increased minute ventilation is necessary to prevent hypercapnia. The development of a subcutaneous or retroperitoneal leak results in the loss of a large quantity of CO_2 from the peritoneal cavity, all of which must be excreted by the lungs because it cannot be expressed from the abdomen after the procedure. We present a patient who developed severe postoperative hypercarbia associated with the development of subcutaneous emphysema from insufflation of CO_2 during laparoscopic cholecystectomy.

SEE THE RELATED EDITORIAL ON PAGE 200 AND THE GRAPHICS CORNER ON PAGE 227.

Case Summary

A 76-year-old woman was initially admitted to the orthopedic service for repair of a fractured femur. Her past medical history was significant for a previous myocardial infarc-

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An abstract based on this case was presented at the RESPIRATORY CARE OPEN FORUM during the 42nd International Convention and Exhibition of the American Association for Respiratory Care in San Diego, California, November 3-6, 1996.

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tion, a 4-vessel coronary artery bypass graft (CABG), congestive heart failure, and a total hysterectomy. The patient also had a pacemaker, placed to treat symptomatic bradycardia following her CABG. Her physical status according to the American Society of Anesthesiologists system was ASA III (indicating a moderate lifestyle limitation and a moderate anesthetic risk with mortality < 5%). She lived alone, functioning independently and caring for her personal needs.

During the patient's recovery from the femur repair, she developed a postoperative ileus. This was believed to be due to pancreatitis as her amylase was markedly elevated. A subsequent ultrasound revealed gallstones. Her diagnosis was most consistent with gallstone pancreatitis. Due to her advanced age and history of cardiac disease, the best course of action was determined to be a laparoscopic cholecystectomy to minimize the postoperative period of immobility by decreasing her pain and need for potent analgesics.

The patient was taken to the operating room and routine monitors (electrocardiograph, automated blood pressure cuff, pulse oximeter) were applied. An intravenous line was placed and general anesthesia was induced with etomidate. Following succinylcholine, her trachea was intubated without complications. A normal CO_2 excretion curve was seen after intubation. Ventilation was adjusted to produce an end-tidal carbon dioxide partial pressure (P_{etCO_2}) of about 38 torr. General anesthesia was maintained with isoflurane, 50% nitrous oxide in oxygen, and intermittent boluses of narcotic (fentanyl). Neuromuscular blockade was continued with pancuronium.

The first trocar was placed with some difficulty due to her previous surgery. After several failed attempts, it was successfully placed and secured with sutures. The surgeon then insufflated the abdomen with CO_2 keeping the intra-abdominal pressure at < 15 cm H_2O. Shortly after the insufflation was begun, the patient's systolic blood pressure dropped below 100 torr. This was treated by the anesthesiologist with a 250-mL bolus of 5% albumin. The patient's arterial blood gas (ABG) results at this time were: pH 7.39, P_{aCO_2} 34 torr, and P_{aO_2} 212 torr. After the fluid load, the patient's blood pressure became stable. As the surgery proceeded the P_{etCO_2} was noted to range from 38-45 torr. Laparoscopic visualization and control of the gall bladder was impeded by adhesions from her previous surgery. Approximately 2 hours into the procedure, the P_{etCO_2} was noted to abruptly rise to 54 torr. Despite a near doubling of the minute ventilation (from 12 L/min to 20 L/min), P_{etCO_2} remained above 50 torr. At that time, the operating team noted that the patient had subcutaneous emphysema throughout her neck and upper chest. Clinical examination revealed no apparent mechanical cause for ventilatory failure (ie, pneumothorax, endotracheal tube dysfunction, or ventilator or circuit problems). The increase of P_{etCO_2} was treated with increases in the ventilator rate. The laparoscopic cholecystectomy was completed, and the patient went to the post-anesthesia care unit.

In the recovery room, an ABG showed: pH = 7.12, P_{aCO_2} = 110 torr, and P_{aO_2} = 92 torr. The patient remained intubated, and mechanical ventilation was continued. Because of the severity of her respiratory acidosis and her past medical problems, the patient was admitted to the surgical intensive care unit (SICU) for management.

In the SICU during the next 2 hours, oxygen concentration was reduced to 50%, and with pressure support ventilation of 10 cm H_2O and 5 cm H_2O of positive end-expiratory pressure (PEEP), an ABG showed pH = 7.49, P_{aCO_2} = 30 torr, and P_{aO_2} = 175. She was extubated, placed on oxygen at 2 L/min by nasal cannula, and monitored overnight. She experienced no arrhythmias, hypertension, or cardiac compromise requiring intervention. The next day, she was transferred to the surgical floor. The rest of her hospital stay was uneventful, and she was discharged home on the second postoperative day.

Discussion

Gas exchange abnormalities seen during laparoscopic cholecystectomy are multifactorial. Abdominal distention compresses posterior lung segments and causes atelectasis. This primarily affects arterial oxygenation by increasing the number of low ventilation-perfusion units and venous admixture. Upper lung segments are relatively overventilated by positive pressure ventilation causing wasted ventilation or increased dead space. The effect of this increased dead space on arterial and P_{etCO_2} should be easily overcome by a small increase in total ventilation. The severe, persistent hypercarbia seen in this patient cannot be explained by increased dead space from positive pressure ventilation alone. It was not responsive to a large increase in minute ventilation.

The adverse hemodynamic effects of pneumoperitoneum and positive pressure ventilation are additive. Increased abdominal pressure decreases venous return, lowering cardiac output and blood pressure.[13,14] In addition to decreasing return to the right heart, positive pressure ventilation further decreases cardiac output by increasing the afterload of the right heart, making it distend into the left ventricle, thus reducing left ventricular stroke volume. The decrease in blood flow through the lung further reduces apical perfusion, increasing dead space volume further and worsening venous admixture. Many of these cardiac effects can be counteracted with adequate fluid loading.

The development of pneumothorax during laparoscopic surgery is a known complication that may have dire consequences.[15,16] Simple pneumothorax can have a profound effect on both oxygenation and CO_2 excretion. A tension pneumothorax can cause cardiovascular collapse. CO_2 is also rapidly absorbed from the lung pleura and can cause a rise in P_{aCO_2} by this mechanism. The use of PEEP to treat a CO_2 pneumothorax occurring during laparoscopic surgery has been described.[17]

The most important cause of the rise in P_{aCO_2} during laparoscopic cholecystectomy is absorption of the insufflated CO_2.

This can occur directly across the peritoneal cavity or from the retroperitoneum or other surface area. Leighton and others[18] produced pneumoperitoneum in swine with both CO_2 and helium. Only with CO_2 was a rise in P_{aCO_2} and P_{etCO_2} observed. Only a slight, clinically unimportant increase in dead space was seen, which did not differ between inflating gases.[19]

Some researchers speculate that the insertion of the insufflating needle into the peritoneum and the abdominal cavity can create a tunnel from the peritoneal cavity to the subcutaneous tissues. This creates a conduit of low resistance, which allows CO_2 to collect and be absorbed.[20] The extent of CO_2 absorption is proportional to the amount insufflated, which depends on the abdominal pressure and the duration of the procedure. From animal studies, this has been estimated to increase CO_2 excretion through the lungs by about 75%.[21]

Considerable quantities of CO_2 can be stored temporarily in muscle and other tissues.[22] The occurrence of subcutaneous emphysema in our patient indicates that a large quantity of CO_2 was insufflated, all of which had to be excreted through the lungs. CO_2 is more rapidly absorbed from subcutaneous tissues than from the peritoneal cavity.[23] The difficulty in placing the insufflation cannula may have been a contributing factor to development of the subcutaneous emphysema.

In light of these possibilities, postoperative care of the laparoscopic cholecystectomy patient should include routine examination of the abdominal and chest wall for the accumulation of subcutaneous emphysema. If subcutaneous emphysema is present, arterial blood should be analyzed to establish the presence of hypercarbia and its extent. A complete patient assessment should be performed to rule out other causes of hypercarbia, such as inadequate ventilation, pulmonary gas embolus, malignant hyperthermia, and pneumothorax.[24]

While the patient's acid-base and hemodynamic status should improve after decompression of the abdominal cavity, CO_2 stored in the tissues still may be entering the bloodstream after insufflation has ended. Hypercarbia and acidosis can cause increased sympathetic nervous system activity, an increase in blood pressure, and cardiac arrhythmias and can lead to CO_2 narcosis if the CO_2 is greater than 100 torr. Hypercarbia and respiratory acidosis can be treated by increasing minute ventilation with the ventilator, but the practitioner must be careful to monitor blood gas values to make sure that respiratory acidosis does not become respiratory alkalosis after the CO_2 load has been excreted.

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Benchmark Data for the Outcome of Mechanical Ventilation in a Pediatric Intensive Care Unit

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"Why do we live? Most of us need the very thing we never ask for. We talk about revolution as if it were peanuts. What we need is some frank thinking and a few revolutions in our own guts; to hell with most of the sons of bitches that I know and myself along with them if I don't take hold of myself and turn about when I need to—or go ahead further if that's the game."

William Carlos Williams
from *Selected Letters of William Carlos Williams*

Benchmarking—What Is It?

Attempts to control the rising cost of health care began in the 1980s with the direct utilization controls and reimbursement restrictions imposed by Medicare and Medicaid diagnosis related groups and per diem payment. The industrial sector has responded with managed care organizations of increasing scope. A major concern of health care providers is that the quality of care may not be as measurable and hence not perceived as important a factor as cost. To address this concern, many health care organizations have been conducting benchmarking studies like those originated by the Xerox corporation in 1973,[1] as a form of outcome tracking.

Benchmarking is a method of improving organizational performance through systematic identification and implementation of best practices.[2-4] Benchmarking usually occurs within the context of comparing similar organizations, but a single organization can effectively establish its own baseline data for documenting the effects of process improvement projects. The Joint Commission on the Accreditation of Healthcare Organizations emphasizes the measurement of outcomes to help determine priorities for improving systems and processes.[5]

Outcome tracking is often focused on activities that involve high cost and/or high risk. Mechanical ventilation of pediatric patients meets both criteria, yet there is a surprising lack of relevant data in the literature.[6-9] The purpose of this study was to describe the outcome of mechanical ventilation in a modern, tertiary-level pediatric intensive care unit (PICU). These data are being used by our institution to identify and document quality improvement. Similar data may be used by other hospitals for the same purpose. To determine best practice, it would be ideal if comparable data from several institutions could be examined. Our data may serve as a start in that direction.

SEE THE RELATED EDITORIALS ON PAGES 202 & 204.

Benchmarking: Mechanical Ventilation in a PICU

Mechanically ventilated children are among the sickest in the PICU and are at high risk for complications. Innovations in ventilator design are introduced continually to meet their needs. At the time of this writing (1996), more than 20 different modes of ventilation are available on at least 24 different models of ventilators, with some models offering as many as 9 modes. Little consensus exists among clinicians about what to call these modes, let alone how to use them. Thus, it is not surprising that there are few validated guidelines and even fewer clinical pathways available for ventilating children with specific diagnoses. Some would argue that premeditated actions are impractical when dealing with complex and physiologically unstable disorders requiring intubation and mechanical ventilation. And, it is unlikely that future controlled clinical trials will uncover clearly superior ventilation strategies. Such controlled trials have generally failed to demonstrate major differences in outcome variables for extremely disparate experimental modes of ventilation (such as high frequency oscillation vs pressure-controlled intermittent mandatory ventilation, or IMV, in neonates) let alone subtle differences in conventional ventilator settings. As a consequence, no major mode of ventilation ever introduced has been entirely abandoned. It is not surprising then, that mechanical ventilation

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remains one aspect of clinical practice that is subject to extremes in practice variation. Yet, current economic forces encourage us to decrease practice variation in order to maintain the best possible outcomes with ever-decreasing lengths of stay and resource utilization.[10] One practical approach to this dilemma is to simply define, track, and report current practice and outcomes as clearly as possible and then compare data among similar health care systems. The details of best practice can then be analyzed, adapted, and incorporated into an evolving body of medical knowledge. This is the central idea of benchmarking.

Materials & Methods

The patients in this study were admitted to the PICU of Rainbow Babies & Childrens Hospital, a 16-bed unit staffed 24 hours/day by 1 attending pediatric intensivist, 1 pediatric critical care fellow, 4 or 5 pediatric residents, 8-16 pediatric nurses, and 2 registered respiratory therapists. Mechanical ventilators in use included the Siemens Servo 900C, the Bird V.I.P., and the Newport Wave E200.

A total of 993 patients who received mechanical ventilation for more than 24 hours in our PICU from 9-7-90 to 2-21-93 were identified. Patients were excluded only if a complete set of data was unavailable. From the 993 patients, a sample of 200 patients were randomly selected. A complete data set could not be obtained on only 2 patients. The remaining 198 patients were compared to the whole patient population (n = 993) in terms of age, gender, and disease type. Student *t* test was used to compare the mean age of our sample and the mean age in the population. The χ^2 goodness-of-fit test was used to compare the distribution of gender and disease in the sample and the population. Statistical significance was decided at the 0.05 level. Data were abstracted from patient charts by clinical nurse specialists and entered into a computer database (Claris FileMaker Pro for the Apple Macintosh computer). Each database record was reviewed for errors and reports were generated using the application's scripting features. Patients who were ventilated more than once (ie, failed one or more extubation attempts) were given separate records for each ventilation period. The study was approved by our Institutional Review Board.

Results

No significant difference was observed between the sample and population mean age. The gender ratio for the sample was not different from that in the population, and the distribution of the disease type in the sample was a good representative of the population disease distribution. Thus, the sample of 198 patients was an appropriate representation of the total patient population, and data from their records were included in the study (Table 1).

Table 1. Demographic Data for 198 Mechanically Ventilated Patients

Diagnostic Category	n (% of total)	Male/ Female	Mean Age in Months (SD)	Mean Weight in Kg (SD)
Cardiac	92 (46)	41/51	22.9 (51.3)	8.6 (12.7)
Pulmonary/ Respiratory	60 (30)	39/21	30.3 (44.6)	11.7 (11.6)
Neurologic	22 (11)	14/8	57.7 (61.1)	21.9 (17.1)
Gastrointestinal	10 (5)	5/5	27.1 (37.7)	10.8 (9.4)
Neurosurgical	7 (4)	4/3	79.6 (58.3)	32.5 (20.4)
Oncology/ Hematology	5 (3)	2/3	164.2 (49.0)	46.0 (18.0)
Renal	2 (1)	1/1	114.5 (102.3)	28.2 (20.6)
All patients		106/92	35.6 (56.2)	13.1 (14.8)

No standardized criteria for intubation were used. Preintubation blood gas values, diagnoses, and subsequent numbers of blood gas samples analyzed are summarized in Table 2.

Synchronized intermittent mandatory ventilation (SIMV) was the mode used almost exclusively. Continuous mandatory ventilation (assist/control) was used 1 time and high-frequency jet ventilation (HFJV) was used 4 times. Pressure support (PS) of spontaneous breaths was used with SIMV 10 times. The mode of ventilation at extubation was usually SIMV (170, or 86% of patients); 17 patients were extubated from continuous

Table 2. Preintubation Arterial Blood Gas Values and Numbers of Samples Analyzed

Disease Category	Mean Preintubation Arterial Blood Gas Values (SD)				Mean Number of Analyses/Patient	Mean Number of Analyses/Patient Day
	pH	P_{CO_2}	P_{O_2}	Base Excess		
Cardiac	7.27 (0.125)	50 (18)	63 (40)	−5.1 (8.1)	54	11.8
Pulmonary/Respiratory	7.24 (0.101)	66 (21)	79 (48)	−1.3 (5.5)	44	5.0
Neurologic	7.12 (0.195)	70 (33)	137 (72)	4.3 (34.9)	27	7.2
Gastrointestinal	7.38 (0.075)	41 (5)	103 (2)	−1.1 (7.1)	41	10.4
Neurosurgical	7.43 (0.001)	24 (1)	324 (220)	−5.1 (0.1)	26	7.0
Oncology/Hematology	7.36 (0.039)	48 (12)	128 (89)	1.3 (7.3)	33	4.5
Renal	7.40 (0.001)	40 (1)	80 (1)	0.1 (0.01)	146	10.0
All patients	7.25 (0.139)	60 (24)	94 (90)	−1.0 (15.6)	47	15

positive airway pressure (CPAP), 6 from SIMV + PS, and 2 from HFJV (both died). The mode of ventilation at extubation was not noted for 10 patients. Ventilator data are summarized in Table 3.

There were 44 episodes of repeat ventilation (ie, first extubation attempt failed). A third ventilation period was required by 8 patients, a fourth by 1, and a fifth by 1 patient.

No specific weaning protocols were followed. The most commonly cited criteria for extubation were adequate blood gases (61%), respiratory rate (51%), and successful CPAP trial (19%) as shown in Table 4. Blood gas values following extubation indicated that patients had returned to normal. Pre-extubation blood gas values for the group (mean, SD) were pH = 7.39 (0.24); P_{aCO_2} = 44 (9) torr, or 5.9 (1.2) kPa; P_{aO_2} = 91 (45) torr, or 12.1 (6.0) kPa, and base excess −2.9 (5.8).

Tidal volumes (V_Ts) were relatively large, with an average of about 12 mL/kg. The largest V_T was 26 mL/kg, and the smallest 5 mL/kg. Frequency distributions for the initial, largest, and pre-extubation V_Ts are shown in Figure 1. The relationships among V_T, diagnosis, and the incidence of volutrauma (ie, pneumothorax, pneumomediastinum, pneumopericardium, or subcutaneous emphysema) are shown in Table 5. The incidence of volutrauma was not clearly related to the size of the V_T, nor did the distributions of V_Ts appear to be related to diagnosis.

Complications were rare (Table 6), with pneumothorax the most common (5% in cardiac patients, 10% in gastrointestinal patients, 9% in neurological patients, 8% in pulmonary patients, 0% in other groups). The time of occurrence for pneumothorax (mean, SD) was 2.8 (4.1) days. Accidental extubation was the next most frequent complication (2% in cardiac patients, 10% in gastrointestinal patients, 7% in pulmonary patients, 0% in other groups) occurring at 7.0 (7.7) days. Nosocomial pneumonia was observed only in cardiac patients (2%). Endotracheal tube obstruction was observed in 2% of cardiac and 5% of neurological patients. (Heated humidifiers with

Table 4. Criteria Used To Determine Readiness for Discontinuance of Mechanical Ventilation and Extubation as Specified in Patients' Charts

Disease Category	Extubation Criterion (% of total patients)*						First Extubation Failed
	CPAP Trial	V_T	NIF	RR	ABG	Unknown	
Cardiac	18	4	0	52	68	20	11
Pulmonary/ Respiratory	20	7	0	50	57	33	23
Neurologic	9	5	5	50	55	27	18
Gastrointestinal	50	0	0	50	50	10	20
Neurosurgical	14	14	0	43	29	29	14
Oncology/ Hematology	0	0	0	20	40	60	0
Renal	50	0	0	100	100	0	50
All patients	19	5	1	51	61	25	16

*CPAP = continuous positive airway pressure; V_T = spontaneous tidal volume; NIF = negative inspiratory force; RR = respiratory rate; ABG = arterial blood gas values.

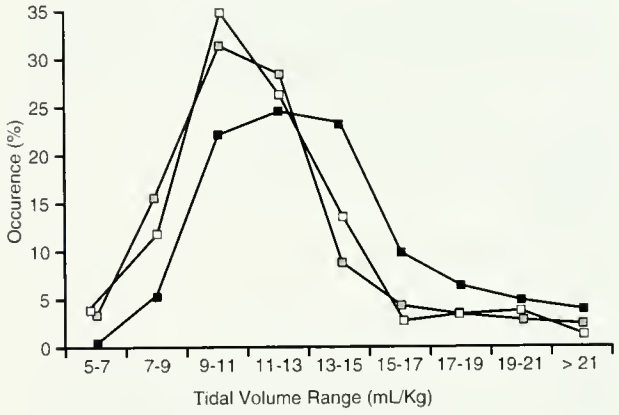

Fig. 1. Frequency distributions for the initial (□), largest (■), and pre-extubation (▣) tidal volumes (summary data for 198 patients).

Table 3. Days of Mechanical Ventilation, Tidal Volumes, Fractional Concentrations of Inspired Oxygen (F_{IO_2}), and Hours at Various F_{IO_2}s

Disease Category	Mean Ventilator Days (SD)	Mean Tidal Volume in mL/Kg (SD)			Initial F_{IO_2} (as % of patients)			Mean Duration of F_{IO_2} Levels in Hours (SD)			
		Initial	Largest	Pre-Ext*	0.40-0.59	0.60-0.79	0.80-1.0	< 0.40	0.40-0.59	0.60-0.79	0.80-1.0
Cardiac	10.6 (35.2)	12.5 (3.2)	14.1 (3.4)	11.9 (3.5)	10	3	77	106 (327)	17 (28)	5 (15)	8 (22)
Pulmonary/ Respiratory	6.2 (6.2)	11.8 (3.4)	14.2 (3.9)	11.8 (3.9)	38	22	30	96 (134)	25 (56)	4 (9)	3 (7)
Neurologic	6.3 (9.4)	10.8 (2.7)	11.2 (2.3)	10.4 (2.2)	41	0	55	95 (131)	8 (14)	4 (9)	3 (4)
Gastrointestinal	5.5 (4.4)	10.4 (1.6)	12.8 (2.8)	11.6 (3.0)	30	10	50	88 (99)	6 (7)	5 (9)	2 (2)
Neurosurgical	4.1 (2.1)	11.8 (2.2)	12.6 (2.4)	11.7 (2.6)	43	0	43	55 (42)	3 (4)	2 (2)	6 (10)
Oncology/ Hematology	5.0 (2.0)	9.3 (0.9)	10.8 (2.6)	8.3 (0.7)	20	20	60	93 (45)	37 (37)	2 (1)	1 (1)
Renal	26.3 (24.8)	13.1 (0.1)	14.9 (0.1)	11.5 (1.5)	0	0	50	514 (494)	9 (3)	0 (0)	1 (1)
All patients	8.3 (24.8)	11.9 (3.2)	13.7 (3.5)	11.6 (3.5)	24	9	57	103 (249)	18 (38)	4 (12)	6 (17)

*Pre-extubation

Table 5. Incidence (Patients with Volutrauma/All Patients in Category) of Pulmonary Volutrauma* as a Function of Tidal Volume and Diagnostic Category

Tidal Volume (mL/Kg)	Cardiac	Pulmonary	Neurologic	Gastrointestinal
5-7	0/4	0/2	0/0	0/0
7-9	0/7	0/8	0/7	0/3
9-11	1/28	0/22	0/6	0/3
11-13	1/30	1/13	1/6	0/2
13-15	0/8	1/6	1/1	0/1
15-17	1/3	1/4	0/2	0/0
17-19	0/3	0/1	0/0	0/1
19-21	2/5	2/2	0/0	1/0
> 21	1/4	2/2	0/0	0/0
	6/92	7/60	2/22	1/10

*Pneumothorax, pneumomediastinum, pneumopericardium, or subcutaneous emphysema.

Table 6. Proportion of Complications and Deaths in Mechanically Ventilated Patients according to Diagnostic Category (n = 198)

Diagnostic Category	Complications* (%)	Deaths (%)
Cardiac	12	18
Pulmonary/Respiratory	15	17
Neurologic	14	27
Gastrointestinal	20	30
Neurosurgical	0	29
Oncology/Hematology	0	100
Renal	0	0
All patients	12	22

*Pneumothorax, pneumomediastinum, pneumopericardium, subcutaneous emphysema, nosocomial pneumonia, ventilator malfunction, accidental extubation, endotracheal tube obstruction.

heated wire patient circuits were used for all patients. Inspiratory temperatures were routinely set at 34° C.) The overall survival rate was 78%.

Discussion

Our study is an attempt to identify critical success factors relevant to pediatric mechanical ventilation and to present data that can be used to compare and improve patient care. There appear to be no similar data in the literature from tertiary-level PICUs in university-affiliated teaching hospitals.

Analysis of our data suggests components of management that could be improved. First, the identification of predictive indices for successful weaning might decrease both the duration of ventilation and the incidence of failed extubation. We have completed preliminary studies of such indices.[11,12] Another factor that might decrease weaning time is the use of pressure support for spontaneous breaths during SIMV.[13] Only about 5% of our patients received SIMV + PS. Our practice now (1996) is to use pressure support with SIMV, as a means both of delivering the breath (ie, higher levels of inspiratory pressure) and of decreasing the resistive work of spontaneous breathing (ie, lower levels).

Although the incidence of volutrauma seems low, it might be decreased even further by using smaller V_Ts, particularly in patients with decreased compliance, like those with acute respiratory distress syndrome (ARDS) or pneumonia. On the other hand, of the 12 patients with volutrauma, none had ARDS (out of 5 patients with ARDS), 4 had pneumonia (out of 25 patients with pneumonia), 1 had AIDS, 1 was a near-drowning, 1 had sepsis, 1 had an esophageal disorder, and the rest had congenital cardiac anomalies. Unfortunately, respiratory system mechanics were not routinely recorded, so it is difficult to judge whether V_Ts and positive end-expiratory pressure were optimized to reduce the risk of volutrauma.

Finally, the number of blood gas assays per patient-day seems high and suggests that costs might be reduced by the application of clinical practice guidelines related to blood gas analysis and noninvasive monitoring.[14] Indeed, a subsequent unpublished study in our PICU suggested that as many as 25% of blood gas analyses may be unnecessary.

Perhaps the most important lesson learned is that we do not have a good system in place to continuously monitor all of the appropriate outcomes. Our hospital information system is based on billing data. We can retrieve information such as total length of stay, total charges, and various cost components, but it is difficult to identify length of stay in a particular unit or nursing division. There is no good way to link particular types of patients to specific outcome variables.

It has become evident that we need to decrease practice variance in the PICU. This can be achieved by designing algorithms that link patient assessment to specific treatment procedures.

These can be in the form of clinical practice guidelines describing particular therapeutic modalities (eg, bronchodilator therapy). Practice guidelines can be combined to form clinical pathways that describe the course of care for particular patient populations (eg, asthma patients). Clinical pathways span the entire length of stay, or even better, the entire circle of care from home, to physician office, to emergency department, to inpatient areas, and back to home. This is the concept of disease management. Practice guidelines and care paths are important not only to reduce practice variation, but as a means to communicate best practices. That is, through the process of benchmarking, one or two hospitals stand out as having better outcomes on some general indicators like length of stay or cost. When this happens, focus groups may be convened to examine the particulars of why these hospitals seem to do better (sometimes called "gap analysis"). More detailed indicator data may be collected and compared among

benchmarking members. At this stage, it should be obvious that clear, specific, and systematic procedures for patient care and documentation are invaluable for making comparative judgments. If every benchmarking partner were able to submit comparable data from similar patient care algorithms, then the strongest algorithm could be identified, emulated, and the process repeated. This is how computer algorithms for chess programs have evolved to the point where they can defeat even the best human players. We have demonstrated a similar phenomenon with computer-managed ventilation of neonates with respiratory distress syndrome.[15] We are not suggesting that computers manage patient care. We are suggesting that systematic procedures be evolved to higher performance levels through a kind of collegial competition in the form of benchmarking.

As we institute guidelines and care paths, we need to track not only macro outcomes, but also algorithm variance data. This provides the vital feedback data that allow us to learn from our experience. Too much of medicine is structured around individual learning rather than team learning. To date, we have had to track these data with custom databases on personal computers in our department. A new hospital information system being installed now should provide the patient order entry and comparative outcome data tracking we need.

In Summary

Our data show that mortality and complication rates were relatively low, with gastrointestinal, pulmonary, and neurologic patients having the highest complication rates. Mortality was not linked to complications nor were complications clearly linked to ventilator management. Mechanical ventilation is safe when administered in a PICU appropriately staffed with well-trained pediatric critical care physicians and respiratory therapists.

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CORRECTION TO AUTHOR INFORMATION

In the paper Manual Volume-Controlled Ventilation in Preterm Neonates Compared to Ventilation via Self-Inflating Resuscitator [Respir Care 1996;41(12):1083-1089] one of the authors, Chris Taylor RRT, was inadvertently listed at Ms Taylor in the author information. It is Mr Chris Taylor RRT, we regret the error.

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Abnormal Capnogram during Laparoscopic Cholecystectomy in a Lung-Transplant Patient

Janice Benedetti CRNA and Charles G Durbin Jr MD

Case Summary

A 53-year-old man presented with a 3-day history of abdominal pain in the right upper quadrant, a white blood cell count of 13,700, a history of gallstones, and an elevated alkaline phosphatase. He was afebrile and in no acute distress, but the right upper quadrant was tender to palpation. His bilirubin was 1.6 IU/mL, and an ultrasound confirmed that multiple gallstones were present without common bile duct dilatation. His past history was remarkable for severe, end-stage emphysema for which he had received a single, right-lung transplant 2 years before this presentation. His pulmonary function has been stable with immunosuppressant medication. Laparoscopic cholecystectomy and common bile duct exploration were scheduled for the next day.

After arrival in the operating room, the patient received general anesthesia and an endotracheal tube was inserted. The patient was chemically paralyzed and mechanically ventilated. He was monitored with capnography during controlled mechanical ventilation (tidal volume = 800 mL, breathing frequency = 6, oxygen concentration = 0.5). Figure 1 shows the capnogram.

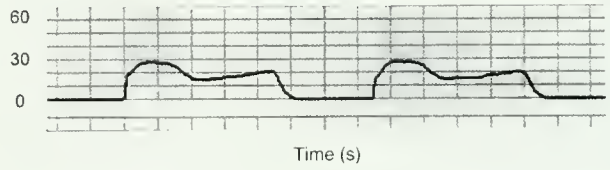

Fig. 1. Postoperative capnogram of patient who received a single-lung transplant 2 years before the present surgery. The interval between each vertical line = 1 second.

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Correspondence/Reprints: Charles G Durbin Jr MD, Department of Anesthesiology, Box 10010, Charlottesville VA 22906-0010.

How would you answer these questions?

Is this a normal capnogram?

If it is not, how do you explain the tracing shown?

Answers and Discussion on page 228

Answers & Analysis

The capnogram is abnormal. The tracing shows a 2-component CO_2 exhalation curve. This is produced by the differences in time constants (due to compliance differences) and in blood flow between the patient's 'floppy' native left lung and his transplanted lung, which has a more normal compliance (ie, stiffer). The first 'hump' is from the less compliant (transplanted) lung's emptying early in exhalation; the second slow rise in CO_2 is from the delayed emptying of the emphysematous lung.

SEE THE RELATED EDITORIAL ON PAGE 200 AND THE CASE REPORT ON PAGE 218.

Differences in expiratory flow pattern after transplantation have been observed following single-lung transplants for emphysema.[1] This abnormality has been thought to be a marker of the development of bronchial obstruction;[2] however, this difference in lung emptying is now recognized as a predictable consequence of single-lung transplantation in patients with severe emphysema.[3,4] Unlike the flow-volume loops described in the literature, capnograms reflect not only the difference in mechanical emptying (due to the more normal elastic recoil properties of the transplanted lung) but also the differences in blood flow to the lungs. This patient's transplanted lung probably received most of his cardiac output, accounting for the high, early peak in the expiratory capnogram. The slowly rising terminal phase is related to the late emptying of areas of the lung with high proportions of ventilation for their blood flow (ie, high V-Q ratios), primarily in the native, emphysematous lung.[5] The patient's preoperative pulmonary function tests showed severe obstruction, with forced expiratory volume in the first second, or FEV_1, of 1.12 L, (predicted 3.48 L); forced vital capacity, or FVC, of 2.16 L (predicted 4.79 L); and FEV_1/FVC of 52%. The patient's expiratory flow-volume loop was diminutive, and the expected biphasic slope was not apparent. However, his peak flow was less depressed (46% of predicted) than was his midflow (11% of predicted).

The surgery was completed in slightly less than 2 hours. The patient was extubated in the operating room without problems and was discharged to his home the next morning, without complications.

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Bronchodilator Response in the 'Borderlands'

Mark Nicolls MD, Jack Wanger RRT RPFT, Charles G Irvin PhD

Case Summary

The patient, a 61-year-old man, presented for evaluation of mild fatigue, exertional breathlessness, and breathlessness associated with bending over. He reported a nonproductive cough without chest pain or hemoptysis. An earlier evaluation had revealed an abnormal chest radiograph, which coupled with symptoms led eventually to an open lung biopsy. The biopsy revealed usual interstitial pneumonitis (UIP), and a diagnosis of interstitial pulmonary fibrosis (IPF) was made. He had been started on prednisone 30 mg a day and cyclophosphamide (an antineoplastic agent sometimes used to treat IPF). The cyclophosphamide had been stopped after 9 months because of anemia, but he has remained on prednisone receiving occasional pulse steroids in response to decrements in pulmonary function or increasing symptoms of exertional breathlessness. Recently, he had undergone thoracoscopic resection of a nodule in the right upper lobe, which proved to be a benign lipomatous lesion. Since that time he had done reasonably well except for an episode of increased dyspnea, which was treated successfully with pulse steroids.

The patient now weighs 103 kg (226 lb) and is 163 cm (64 in.) in height. He uses oxygen by nasal cannula at 2 L/min. His respiratory pattern is normal with equal bilateral expansion of the chest wall. Percussion is normal. Auscultation reveals subtly increased fremitus at the bases bilaterally. Further, he has scattered wheezes diffusely on forced expiration and bilateral focal end-inspiratory crackles. The expiratory phase of respiration is mildly prolonged. The chest radiograph taken during this examination is shown in Figure 1 and demonstrates mildly diminished lung volumes, predominantly peripheral reticular opacification, and diffuse bronchial wall thickening. Results of pulmonary function tests are shown in Table 1 and Figure 2.

Fig. 1. Chest radiograph (posteroanterior) of 61-year-old man with a diagnosis of interstitial pulmonary fibrosis.

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Table 1. Results of Pulmonary Function Tests Administered to a 61-Year-Old Man with a Diagnosis of Interstitial Pulmonary Fibrosis

Variable*	Before Bronchodilator		After Bronchodilator	% Change
TLC (L)	3.74	(76)†	3.76	1
RV (L)	1.56	(108)	1.45	−7
$FRC_{plethys}$ (L)	1.99	(60)	1.81	−4
FVC (L)	1.74	(46)	1.83	5
FEV_1 (L)	1.23	(47)	1.38	12
FEV_1/FVC (%)	71	(103)	75	6
sGaw ($L \cdot s^{-1} \cdot cm\ H_2O^{-1}$)	0.16	(84)	0.16	0
DLCO ($mL \cdot min^{-1} \cdot torr^{-1}$)	15.8	(56)	—	—
V_A BTPS (L)	2.98	(53)	—	—
$DLCO/V_A$	5.28	(105)	—	—
PI_{max} (cm H_2O)	60	(71-135)	—	—
PE_{max} (cm H_2O)†	155	(135-283)	—	—

*Abbreviations are identified in the text except for PI_{max} = maximum inspiratory pressure; PE_{max} = maximum expiratory pressure.
†Values in parentheses are percent of predicted.

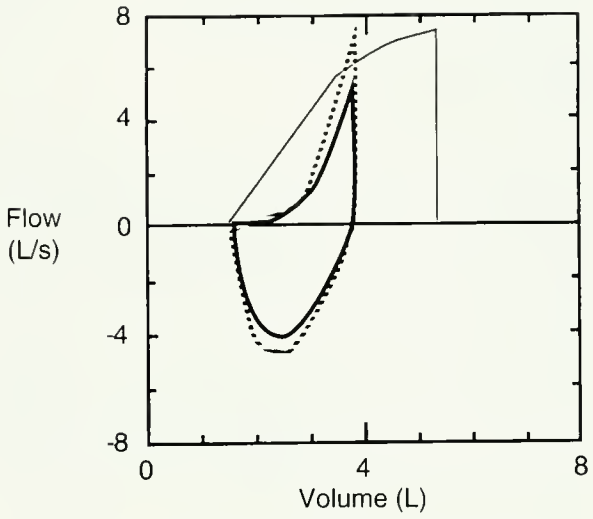

Fig. 2. Flow-volume loops before and after bronchodilator plotted at absolute lung volume for a 61-year-old man with a diagnosis of interstitial pulmonary fibrosis.

How would you answer these questions?

1. How do you interpret the results of these pulmonary function tests?

2. Do these tests meet the necessary criteria to be considered positive for bronchodilator response?

3. Do you recommend that this patient be given a bronchodilator?

Answers and Discussion appear on page 232

Answers & Discussion

Test Interpretation

The total lung capacity (TLC), functional residual capacity measured by body plethysmograph ($FRC_{plethys}$), volume exhaled in the first second of forced expiration (FEV_1), and forced vital capacity (FVC) are all markedly reduced. The FEV_1/FVC is slightly higher than 100% of predicted, suggesting a restrictive process.[1,2] The patient's obesity may be a factor. The weight-in-kilograms to height-in-centimeters ratio of 0.63 could account for a low FRC but cannot explain the low TLC and carbon monoxide diffusing capacity (DLCO).[3] The DLCO is markedly reduced but corrects into the normal range when adjusted for alveolar volume (V_A), which is frequently the case in interstitial lung diseases.[4] Pure restrictive disease is rare, and pathologic examination usually reveals varying degrees of airways involvement. Mixed disease is relatively common, and these patients are frequently treated with bronchodilators. This patient probably has a mixed process because flows at isovolume are not elevated, the FEV_1/FVC is not grossly elevated, the residual volume (RV) is not reduced, and there is a suggestion of a bronchodilator response. In light of the patient's known clinical history, restrictive lung disease with an element of airways disease is an appropriate diagnosis.

Bronchodilator Response

A bronchodilator treatment (0.5 mL of albuterol) was administered in the laboratory and resulted in a small increase in heart rate. After completion of the treatment and a 15-minute waiting period, spirometry, specific conductance (sGaw), and lung volumes measurements were repeated. The results are shown in Table 1.

The American Thoracic Society (ATS) criteria for significant bronchodilator response is based on a synthesis of findings from studies conducted in the 1970s and 1980s that utilized both healthy volunteers and patients. These guidelines state that "a 12% increase, calculated from the pre-bronchodilator value, and a 200-mL increase in either FVC or FEV_1 are reasonable criteria for a positive bronchodilator response in adults."[2] This guideline represents the consensus opinion of a panel of experts, but debate over its sensitivity and specificity continues. As reviewed by Hansen et al,[5] depending on the study, values from 8-25% improvement in FEV_1 or 10-15% improvement in FVC have been offered as levels of significant change. Placebo testing can often be useful in distinguishing true treatment responses, but the act of performing spirometry in and of itself without a bronchodilator can induce changes in pulmonary function tests.[6,7] Individual variability adds to the confusion. As with any response to a given stimuli, an upper range of normal variability must be established—beyond which a real response can be said to occur. The principal problem in establishing an upper range in this clinical situation is that the range varies depending on the population studied and the techniques employed.[8]

This patient met the 12% criterion for change in FEV_1 but failed to make the 200-mL volume-change criterion. Thus, by ATS criteria, the bronchodilator response is not considered to be positive. Unfortunately, this patient might get true benefit from a bronchodilator because his respiratory reserve is so low. In a patient with a large baseline FEV_1, an increase of > 200 mL could be of relatively smaller importance than the 150-mL change noted in this ailing patient. If one looks beyond the FVC and FEV_1 in this patient, it appears that there is little suggestion of a bronchodilator response. The sGaw and lung volumes were mostly unchanged, although one could argue that the 7% reduction in RV suggests a response.

Decision To Treat

An important caveat in the ATS guideline is the statement "a patient may respond to long-term bronchodilator therapy even though a bronchodilator response is not seen in a single laboratory testing session."[2] In other words, a negative response in the laboratory by the above criteria does not preclude a benefit from therapy. It is the responsibility of the caring physician to weigh the risks and benefits of bronchodilator therapy (as with any therapy) and make a determination about the decision to treat. In this patient, it was determined that the possible benefit of treatment outweighed possible disadvantages, such as cost and drug side effects. A bronchodilator was prescribed for the patient.

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Books, References, and Directories Now Available from the Publishers

Anyone Can Intubate: A Practical, Step-By-Step Guide for Health Professionals, 4th Edition, by CE Whitten MD. Softcover, 240 pages, illustrated. San Diego: KWPublications, 1997. $19.95. Available from the publisher at (800) 450-2665. [Respir Care 1994;39(10):1002-1003]

Children with Asthma: A Manual for Parents, by TF Plaut MD. South Deerfield MA: Pedipress Inc, 1995. The pocket-sized edition is available for $10 each or $80 for 10. Other patient education materials are available including **One-Minute Asthma: What You Need to Know**, call (800) 611-6081.

Statistics for the Social Sciences, by RM Sirkin. Softcover, 502 pages. Thousand Oaks CA: Sage Publications Inc, 1995. $35.

Upon opening Mark Sirkin's **Statistics for the Social Sciences**, I initially focused on what was missing from the book. I would have expected a book for social scientists to pay considerable attention to statistical models that use multiple independent and dependent variables such as discriminant analysis, canonical correlation, factor analysis, multivariate analysis of variance, and multiple regression. Sirkin covers only multiple regression.

However, Sirkin wrote his book for a different audience; and, for them, it is a book with great utility. From the Preface, the book "is designed to teach introductory statistics primarily to undergraduates." Because one must walk before learning to run, I adjusted my expectations and proceeded. I also found that Sirkin and I share instructional philosophies. Sirkin stresses to students some of the same points I have expressed: Don't try to "cram" the material; this course will take regular sustained attention; ask for help when you feel overwhelmed; and attend class! Basic stuff—but important nonetheless.

In the first chapter, "How We Reason," Sirkin creates a link to the scientific method—a link missing from many statistics texts. Sirkin points out that we seek facts rather than values with statistics and proceeds to define scientists, hypotheses (inductive and deductive), data, independent and dependent variables, association and causation. Then I found a statement I was most pleased to find up front.

> (Using statistics) we seek to generalize from a smaller group (the sample) to a larger group (the population), but can never be absolutely certain that the findings from the sample are true for the population. Instead we use statistics to estimate the probability that the findings hold for the population.

Sirkin's book explains the reasons for using statistical tests—a positive dimension, particularly if one is selecting a book to introduce statistics to respiratory care students. I guess that most instructors prefer a text that uses medical examples. However, the examples Sirkin uses to illustrate his points relate well to a typical undergraduate's life experience and may better facilitate understanding of statistical concepts.

Sirkin also gives ample exercises after each chapter with answers and explanations at the end of the book.

Sirkin provides practical, easily understood examples of nominal, ordinal, interval, and ratio levels of measurement. A statement telling the reader that levels of measurement often drive the kind of research and the statistical test one can apply to the data would be a nice addition. However, such a statement appears later in the book.

Because cognitive measurement is now my career, I am particularly interested in Sirkin's treatment of validity and reliability in the third chapter. In "Defining Variables" he addresses the importance of pilot testing, content, criterion, and construct validity—all in 4 pages. One will need to look elsewhere for more detailed explanations.

"Central Tendency" covers predictable content (eg, mean, median, and mode) but also explains commonly used abstract symbols (eg, Σ, n, $\times_i$, μ, σ), and gives examples of frequency distributions, stem and leaf displays, and box plots.

"Measuring Dispersion" provides a clear explanation of the relationship between variance (S^2) and standard deviation (S) and introduces the reader to the fact that many statistical concepts have definitional and computational formulas.

In "Constructing and Interpreting Contingency Tables," Sirkin gives practical advice including guidelines for creating ordinal level groups from interval or ratio level data.

"Statistical Inference and Tests of Significance" introduces the reader to all the inferential statistics and discusses null and alternative-research hypotheses, and introduces the *t* tests and analysis of variance as models for comparing means. The author lays out 9 sequential steps for applying a significance test including the null hypothesis statement, collecting data, calculating the statistic, stating the p value for Type I error, and then stating the conclusion—a nice checklist for novice or experienced researchers.

In "Probability Distributions and One-Sample *z* and *t* Tests," Sirkin describes the

characteristics of normal distributions. Sirkin rewrites the *z* formula in clear terms. He then addresses the normality assumption and stresses the importance of using large sample sizes to better ensure that the assumption is met. He also provides a table showing varying sample sizes and tells the reader to what degree it is safe to relax the normality burden.

Within the chapter titled, "Two-Sample *t* Tests" is another discussion of directional hypotheses including when it is appropriate to pose such statements. The reader is also introduced to the discipline of checking to see if the assumptions (eg, homogeneity of variances) underlying a statistical test are met.

This chapter ends with a clear explanation of the statistical power concept (although the computation of power was presumably beyond the scope of this book) indicating simply that it derives from the difference between group means and sample size.

In "One-Way Analysis of Variance," Sirkin introduces the notion of doing both the initial ANOVA and a post-hoc test (we already know that the test is used to compare means with more than 2 groups). Rather than jump right into the mechanics of ANOVA, Sirkin takes care to specifically cover terminology such as sum of squares, within-groups and between-groups, sum of squares, and mean square.

Though I initially lamented the lack of discussion on multivariate statistical models, Sirkin's book does lay a good foundation for later study. For example, when addressing post-hoc testing, he points out that it is best to avoid running multiple *t* tests because a Type I error would inflate with each comparison (a primary reason multivariate procedures exist).

Like many other texts, Sirkin offers Scheffé's Test for post-hoc ANOVA work. However, he doesn't address the fact that the conservatism of Scheffé's Test can lead to the untenable situation whereby the ANOVA F test suggests rejecting the null hypothesis while Scheffé's Test finds no differences between groups. While he acknowledges other post-hoc tests, he does not recommend one. I am sure undergraduates would appreciate a recommendation.

"Measuring Association in Contingency Tables," Sirkin covers the implications of measurement scales (eg, nominal, ordinal) on the kind of statistic one can apply. Again, he offers helpful advice to students about constructing contingency tables to present information from 2 dichotomous variables.

In the chapter "The Chi-Square Test," Sirkin gives pronunciation guides to the Greek symbols used to represent some statistical tests and clearly explains the important distinction between research and statistical significance. Sirkin also makes the critical point that no single significant finding is as useful as repeating the study and finding similar results.

In the final chapters, Sirkin covers correlation and multiple regression, describes when to use correlation-regression, and asserts that the value r^2 gives us the proportion of variation in the dependent variable explained by variation in the independent variable.

Sirkin clearly makes what I consider to be the most important point about correlation-regression: The coefficient of determination (r^2) helps "keep us honest when we feel the urge to inflate the importance of our findings." Sirkin carries this theme to the very end of his book asserting that statistics is primarily about applying the rules of logic and that even when a finding appears statistically significant, there still exists at least some probability that the finding was wrong.

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Pulmonary and Critical Care Pharmacology and Therapeutics, by AR Leff, editor (107 contributors). Hardcover, 1,219 pages. New York: McGraw-Hill, 1996. $150.

To read and understand the 119 chapters of this encyclopedic work requires not only a comprehensive background in general pharmacology, but also a familiarity with the terminology of molecular biology, immunology, and biochemistry. Based upon information drawn from these fields, the editor has assembled in-depth discussions of most pertinent subjects relating to the use of drugs in pulmonary medicine and critical care.

A work of this magnitude has not appeared for about 20 years, during which time there has been an enormous output of basic research concerning the lungs and airways. Fully a third of the book is devoted to the properties of bronchopulmonary cells, their morphology, receptor sites, and biochemical responses to endogenous transmitter and mediator compounds. The individual drug families are introduced with reference to the molecular changes induced at the cellular level. It is on these fundamental concepts that the subsequent sections on clinical therapeutics are constructed.

The book is organized in 7 parts; the first 2 are devoted to basic pharmacology, the latter 5 mainly to clinical application. Part 1, Principles of Pulmonary Pharmacology, consists of 40 chapters covering bronchopulmonary cell properties and related neurohumoral and immunologic functions. The chapters generally are brief and concise, allowing the uninitiated reader to focus on a single facet of the cytochemical picture at a time. The first section opens with 3 chapters on receptor types, an appropriate introductory analysis of macromolecular substances that react to the intrinsic compounds that form the basis of drug design. The following sections deal with autonomic innervation of the respiratory system, mediators of inflammation, airway smooth muscle regulation, and the nature and physiologic roles of cytokines. This leads to a general overview of inflammatory mechanisms in the lungs and airways. The final section reviews external toxic substances and other environmental factors that cause respiratory disease.

Part 2 is Pharmacology of Conducting Airways. It is a 250-page compendium covering most of the topics of respiratory pharmacology essential to the core curriculum of a respiratory care program. The first 3 sections are about acute bronchodilator compounds: β-adrenergic agonists, phosphodiesterase inhibitors, and anticholinergic drugs. Then, corticosteroids and other anti-inflammatory therapies are considered, along with newer anti-asthmatic agents directed against specific metabolites such as leukotrienes and thromboxanes. Antitussives and mucolytics are given ample coverage, and a section on adverse drug reactions completes the review of basic pharmacology.

The clinical portion of the work is, to a considerable degree, a series of applications of the basic concepts presented in Parts 1 and 2. Of primary interest is the treatment of obstructive lung diseases (Part 3), which occupies about 200 pages and presents considerable new data on the subject of asthma. Chapters on the diagnosis, genet-

ics, and epidemiology of asthma lead to the central theme of selecting a treatment regimen. New advances in therapy are presented, including the use of cytokines, immunosuppressive agents, and other compounds designed to antagonize bronchoconstrictor peptides and eicosanoids. Of particular interest to specialists in emergency medicine is a chapter on the pharmacotherapy of status asthmaticus.

A section on the treatment of chronic obstructive pulmonary disease follows, in which both standard and innovative regimens are described, including the use of oxygen. Physiotherapy and rehabilitative procedures are reviewed. A brief section on antibiotics for respiratory tract infections is included, along with a similar chapter dealing with cystic fibrosis patients. The section on cystic fibrosis presents chapters on the genetics of the disease and new developments in gene therapy. Bronchiectasis, covering diagnosis and management, is the subject of an informative chapter.

Part 4 is a section on pulmonary embolism, its natural history, diagnosis, and treatment, and a discussion of its most common cause, deep venous thrombosis. Part 5 comprises the various treatments for systemic critical illness where the lungs are involved, including 3 chapters on the acute respiratory distress syndrome. Pulmonary infections accompanying sepsis and multisystem organ dysfunction, particularly in immunodeficient patients, are discussed fully in 4 chapters. The evaluation and treatment of sleep apnea and other hypoventilation syndromes are other topics covered in this section.

Part 6 contains chapters on idiopathic parenchymal lung diseases (fibrosis, connective tissue disorders, and sarcoidosis) and rare disorders of the lung (pulmonary hypertension, hemosiderosis, Goodpasture's syndrome, and Wegener's granulomatosis). The final section (Part 7) includes an analysis of aerosol therapy, a discussion of lung transplantation, and the management of bronchospasm during anesthesia and the perioperative period. There are no chapters dealing with the chemotherapy of bronchopulmonary neoplasms.

This is an excellent reference work. The chapters are well documented, providing the reader with a comprehensive historical background on nearly all subjects. The 52-page index provides specific and detailed entries, which facilitate cross-referencing among related topics. The preliminary sections although esoteric to the average clinical practitioner, represent a rational point of departure toward the recent developments in therapeutics, and provide a continuum of scientific knowledge from the fundamental discoveries to state-of-the-art respiratory care practice.

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Letters

Letters addressing topics of current interest or material in RESPIRATORY CARE will be considered for publication. The Editors may accept or decline a letter or edit without changing the author's views. The content of letters as published may simply reflect the author's opinion or interpretation of information—not standard practice or the Journal's recommendation. Authors of criticized material will have the opportunity to reply in print. No anonymous letters can be published. Type letter double-spaced, mark it "For Publication," and mail it to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

A Problem with Bear Cubs and Isothermal Circuits?

While performing pre-use checks* on Bear Cubs*, I have observed what I believe to be a previously unreported problem that results in a decreased inspiratory time (T_I), with no change in set or displayed inspiratory time, when Isothermal's™ neonatal and pediatric circuits (assembled of corrugated tubing) are used and the Cub's flow is set at 15-30 L/min.

The probability of occurrence of the problem appears to be greatest at the highest flow setting and lowest positive end-expiratory pressure (PEEP) setting. The exact flow at which the problem first manifests is specific to the individual Bear Cub, the particular circuit, and the PEEP setting. The only reliable sign is a 'twitch' in the pressure gauge at the onset of inspiration.

The decrease in actual T_I causes an unexpected drop in peak inspiratory pressure, or PIP, when the decreased T_I exceeds the pressure plateau time. At higher flows or shorter T_Is, the actual PIP can decrease to any degree. At the highest flows, the delay in expiratory valve closure exceeds the set T_I with failure to cycle and activation of the "vent-inop" alarm.

A sharp unexpected rise in PIP occurs if the flow is reduced and the PIP control has been previously altered to compensate for a decreased PIP caused by a decreased actual T_I.

I have been able to reproduce this problem with the 3 Bear Cubs that we own and all rented Cubs. I have no experience with smooth-bore disposable circuits or other manufacturers' disposable neonatal or pediatric circuits.

To avoid problems when aerosol treatments are administered in line with the Cub, I've found it useful to decrease the Cub's set flow by the same amount as the flow driving the nebulizer.

I encourage the manufacturer of Isothermal circuits to alter the labeling of the circuits to reflect flow limitations with the Bear Cub and to design disposable circuits that permit the Bear Cub to be used safely to full capacity.

Michael S Lasky RPFT RRT
Perinatal Pediatric Practitioner
Staten Island University Hospital
North Staten Island, New York

Mr Lawson of Allied Healthcare Products responds:

As the manufacturer of the Bear Cub Ventilator, we agree with Mr Lasky's finding. Similar findings were reported in a Clinical Application Bulletin (CAB), Number 9, published in November 1992. In that CAB we stated:

> The BEAR CUB Ventilator employs a servo-operated pneumatic control system which uses the Proximal Airway line as the path of the feedback loop ... the BEAR CUB exhalation valve includes a jet pump which was specifically designed with the 'standard' patient circuit described in the Instruction Manual. Variations in patient circuit characteristics may result in pressure drops which exceed the capacity of the jet pump. If this occurs, it may imbalance the servo loop.

The problem reported by Mr Lasky is the result of a patient circuit with excessive resistance at the reported flows. In CAB #9, we also outlined a procedure the clinician can use to determine the flow resistance in the expiratory limb of the patient circuit of the Bear Cub Ventilator. Depending on which version is used, the Bear Cub Ventilator can compensate up to 4-5 cm H_2O. Mr Lasky's circuit-flow combination appears to produce an excessive amount of resistance. As a result, there is an increase in inadvertent PEEP which causes the servo system to become imbalanced and results in premature opening of the exhalation valve (decreasing inspiratory time and peak inspiratory pressure).

Because Isothermal circuits (Allegiance Healthcare Corp, Isothermal Systems, Riverside CA) are custom designed for individual hospitals, as are many other disposable ventilator circuits, we developed a Circuit Compensate Chart (Fig. 2, CAB #9), which allows every hospital to establish the 'acceptable' working flow range for the ventilator-circuit system being used by the hospital. We recommend that every hospital thoroughly test all disposable circuits prior to ordering them from suppliers. Each circuit has a different resistance, and most ventilators have different compensation thresholds, which could adversely affect the performance of the ventilator.

As a responsible manufacturer, we publish CABs for all registered owners of Bear Ventilators when situations similar to the one reported by Mr Lasky are discovered. If a hospital failed to receive CAB #9 and would like to be receive future bulletins, please contact me at 909 351-4845.

Damon R Lawson MBA RRT
Marketing Manager
Allied Healthcare Products Inc
Riverside, California

and Mr Uhlemann and Mr Pierro add:

We have performed tests in our laboratory using a Bear Cub Ventilator and the infant circuit that Mr Lasky used in his tests and were able to confirm his findings on the infant circuit but could not recreate the situation on a standard pediatric circuit.

Mr Lasky suggests that Isothermal should alter labeling to reflect circuit flow limitations with the Bear Cub. The current labeling contains the following caution:

> Be aware that compliance and flow resistance characteristics of the circuit may affect the operating parameters and capabilities of the breathing machine.

This caution is placed on the label to address the concerns raised by Mr Lasky.

However, we do not make claims for a particular circuit because it depends on the entire system, including the ventilator, humidifier, and additional equipment. Therefore, we believe that our labeling is appropriate.

We configure Isothermal circuits to customer specifications. Samples are sent to customers for their evaluation. Feedback from the customer to our sales force of design engineering department confirms that the design meets the customer's requirements.

We encourage our customers to perform all the necessary checks, as indicated by the ventilator manufacturer. If there are questions on the compatibility or performance of Isothermal circuits, the customer should contact their Allegiance Sales Representative or call our Customer Service Department at 1-800-321-3832.

Rick Uhlemann
Quality Manager
Respiratory Therapy

Brian Pierro
Senior Principal Engineer
R & D, Respiratory Therapy

Allegiance Healthcare Corporation
Riverside, California

Observations on the Therapist's Role

I congratulate Ms Kirby and Dr Durbin on the findings reported in "Establishment of a Respiratory Assessment Team Is Associated with Decreased Mortality in Patients Re-Admitted to the ICU," which appeared as a special article in the October 1996 issue of the Journal.[1] Their conclusion that a "dedicated respiratory therapy assessment team" reduces mortality in patients re-admitted to the intensive care unit (ICU) underscores the importance not only of respiratory therapists but also of the care that they deliver outside of the ICU.

I believe that further study needs to be focused on our role and possible value in reducing mortality, morbidity, and cost. One such study appeared in *Critical Care Medicine* in 1992.[2] The authors documented the value of the respiratory care practitioner in pediatric transport and in airway management. The authors assert, "The presence of a trained pediatric respiratory therapist contributed to the reduction of the complication rate." We need more demonstration and quantitation of the importance of respiratory therapists, particularly as hospitals downsize and managed care takes root.

I have observed that we as respiratory therapists often underestimate the importance of 'routine' or so-called floor care. We need to stop thinking that providing care for patients 'on the floors' is less important than caring for patients in the ICUs. If we can reduce the death rate in patients re-admitted to the ICU as demonstrated by Kirby and Durbin and, better yet, provide the care that eliminates the need for re-admission, then the value of floor care is obvious. By logical extension, it can be argued that respiratory therapists should fight 'tooth and nail' to keep responsibility for basic floor care.

Greg Hagan MAE MA RRT
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2. Beyer III AJ, Land G, Zaritsky A. Nonphysician transport of intubated pediatric patients: a system evaluation. Crit Care Med 1992;20(7):961-966.

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Contributions to this section are welcome. Send double-spaced typed material (and illustrations if pertinent) to Historical Notes, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593. Identify source of material and include your name, credentials, and address.

Effective Inhalation Therapy: Bronchial Asthma

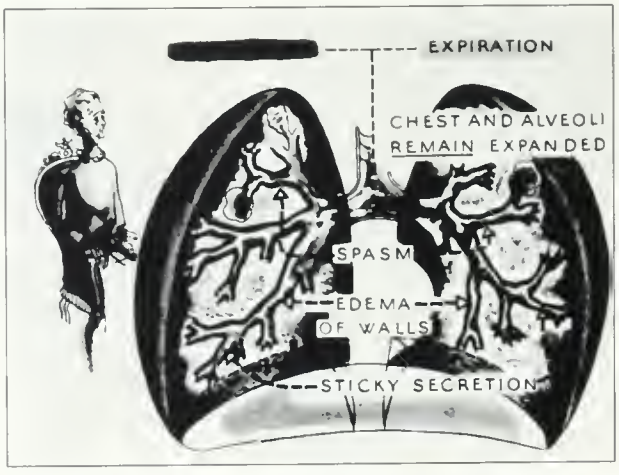
This is a condition in which inhalation therapy is of great value. The chief characteristic of asthma is difficulty in breathing. Long before the acute seizures, the patient feels a heaviness of the chest, which is followed first by the sensation and then the noise of respiratory wheezing. As the disturbance progresses, both inspiration and expiration become increasingly difficult. The wheeze is louder and the dyspnea of the patient is worse. Accompanying all the stages is a state of apprehension and anxiety, which frequently becomes panic as the sensation of shutting off of air occurs.

The changes occurring in the bronchi and lungs explain the symptoms. There is an increasing contraction of the bronchial musculature, which does not relax in the customary fashion during inspiration. In addition, there is an edema and swelling of the mucous membrane and secretion, the constant accompaniment of bronchial irritation. In this case the secretion is generally white and very sticky.

Almost every asthmatic who has had this condition for a period of time develops some bronchial infection. This, producing the changes associated with bronchitis, adds to the trouble. The difficulty in breathing, the back pressure and infection produces the secondary lung changes of pneumonitis, fibrosis and emphysema. Consequently, the difficulties which an asthmatic patient shows are complex and of many different causes. From the bronchial standpoint there is the narrowing of the bronchus by contraction and by swelling of the thick membrane. Added to this is the partial obliteration of the lumen by the thick secretion. These two factors together interfere very seriously with the ventilation of the lung. The greatest interference is in expiration, and the volume of air in the lungs builds up until the chest remains expanded, and even if air could be introduced during inspiration, there would be nowhere for it to go.

Consequently, in severe attacks, particularly those in status asthmaticus, the violent and rapid respiratory efforts accomplish very little in the way of actual ventilation of the lung. In such a state it is necessary to do three things; keep the bronchi open, ventilate the alveoli, and get oxygen into the alveoli. Inhalation therapy is essential to accomplish this. Antispasmodic drugs may be used as aerosols, but more important than that is the use of a pressure mask and helium mixtures.

A positive pressure mask or helmet which maintains a measured degree of pressure on expiration helps to prevent the closure of the bronchi. In addition, it tends to decrease some of the edematous reaction. With such a mask in severe states, a mixture of helium and oxygen should be used. Helium by reason of its light mass will penetrate small apertures and thus enter bronchi that oxygen or air alone would have difficulty entering. It will carry oxygen with it and re-establish normal ventilation. Although the volume of helium present is what makes ventilation more efficient, only the oxygen reaching the parenchyma is used in respiration. Therefore, the percentage of oxygen should never be less than 25 and in very dyspneic patients it would be better to use 35 to 40 per cent. As the condition improves, the helium mixture may be replaced by oxy-



The triple cause of difficulty in breathing in bronchial asthma; sticky secretion, edema of the bronchial walls, and spasm. In expiration the obstruction is greater and air cannot leave the chest.

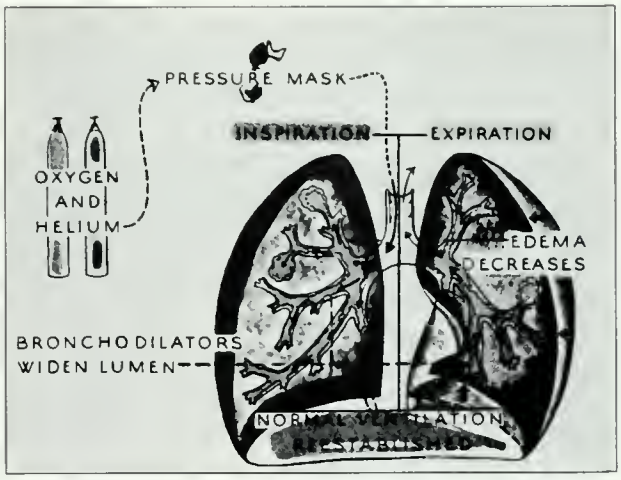
From Effective Inhalation Therapy, written by Edwin R Levine MD in cooperation with Alvan L Barach MD, J Winthrop Peabody MD, and Maurice Segal MD, and published in 1953 by the National Cylinder Gas Co of Chicago. Dr Levine, a chest physician, was an early champion of our profession and President of the American Association for Inhalation Therapy in 1952 (the only physician in the Association's history to so serve). The other authors were also pioneers in pulmonary medicine and inhalation therapy.

This excerpt was made available to RESPIRATORY CARE by Teri Nikolai Wilson RRT RPFT, United Healthcare of Ohio, Centerville, Ohio.

gen. Then the positive pressure mask will continue to be an advantage until respiration is easy.

Even as these mixtures improve severe asthmatic attacks, inhalation therapy may prevent the development of symptoms. The feeling of apprehension and fear which is always present in asthma tends to accentuate the changes in the bronchi that causes the trouble. This fear is based upon the difficulty in breathing. If oxygen is used intermittently early enough when breathing is still relatively easy, the attacks may be prevented. As the patient becomes more apprehensive, he will breathe faster with more effort. These actions cause increased bronchial spasms and more trouble. There is some evidence also that anxiety tends to produce the bronchial changes that lead to asthmatic attacks.

If at this time the patient is given oxygen in adequate percentages, he will find it possible to breathe more easily. Even a few breaths like this may be sufficient to relieve his fear and enable him to relax his bronchi, thus breaking the progression of more rapid respiration, more bronchial spasms, more edema, more trapping of air, and more and more symptoms. If this is done, it is sometimes possible to avoid the use of drugs to dilate the bronchi. This is a matter of some importance, since asthmatic patients generally receive too much medication and become resistant to valuable drugs. In long standing asthmatic patients, the dyspnea and weakness will be very much worse in relation to the amount of bronchial disturbance that is found in more recent cases. This may be explained by the lung changes mentioned above which occur as asthma becomes chronic.



Inhalation therapy provides ventilation through physical means, relief of costriction by aerosols, and improvement in the pathology as well as the condition of the patient.

Consequently, two things must be remembered: first, that there may be difficulty in gas exchange in the lung parenchyma and a higher oxygen circulation will be needed to insure adequate saturation of the hemoglobin; and secondly, that these patients may fall into the category of the chronic anoxic individual in whom it is dangerous to increase the oxygen intake too rapidly. This is a matter that requires some judgment and should be handled according to the suggestions made in the section on chronic anoxia.

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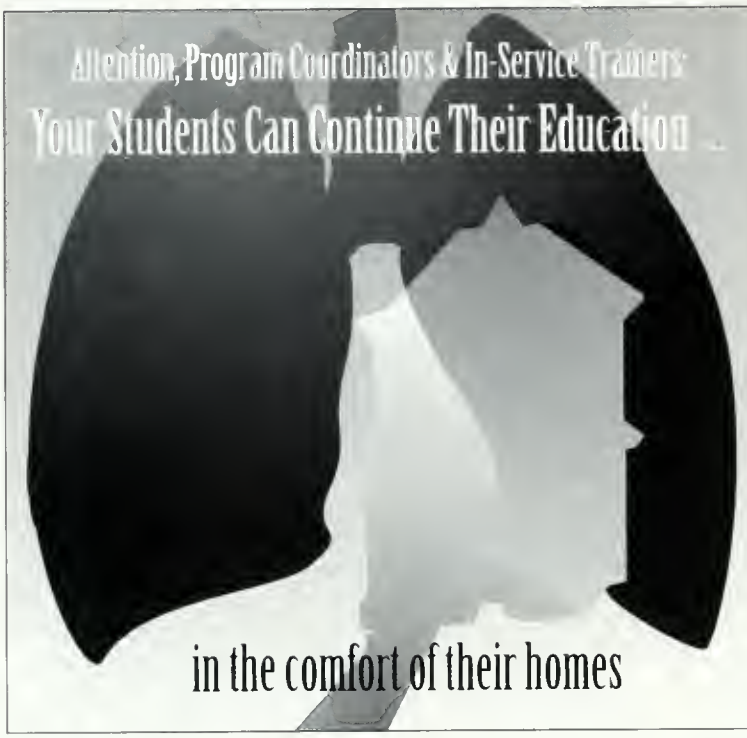
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The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Convention and Exhibition in New Orleans, Louisiana, December 6-9, 1997. Accepted abstracts will be published in the November 1997 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation.

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An abstract may report **(1) an original study, (2) the evaluation of a method, device or protocol, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract is the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocol evaluation. Abstract *must* include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: Relevant basic information important to understanding the case. (2) Case Summary: Patient data and response, details of interventions. (3) Discussion: Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

Accepted abstracts will be photographed and reduced by 40%; therefore, the size of the original text should be *at least* 10 points. A font like Helvetica or Geneva makes the clearest reproduction. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location; underline presenter's name. Type or electronically print the abstract *single spaced in a single paragraph in the space provided on the abstract blank.* Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. Data may be submitted in table form, *and simple figures may be included provided they fit within the space allotted.* No figures, illustrations, or tables are to be attached to the abstract form. Provide all author information requested. A clear photocopy of the abstract form may be used. Standard abbreviations may be employed without explanation; new or infrequently used abbreviations should be spelled out on first use. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (972) 406-4667.

Deadline Allowing Revision

Authors may choose to submit abstracts early. Abstracts postmarked by March 17, 1997 will be reviewed and the authors notified *by letter only* to be mailed by April 25, 1997. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the final deadline (May 27, 1997).

Final Deadline

The mandatory Final Deadline is May 27, 1997 (postmark). Authors will be notified of acceptance or rejection *by letter only*. These letters will be mailed by August 15, 1997.

Mailing Instructions

Mail (Do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

RESPIRATORY CARE OPEN FORUM
11030 Ables Lane
Dallas TX 75229-4593

1997 Respiratory Care Open Forum

Abstract Form

18.8 cm or 7.4"

13.9 cm or 5.5"

1. Title must be in all upper case (capital) letters, authors' full names and text in upper and lower case.
2. Follow title with all authors' names including credentials (underline presenter's name), institution, and location.
3. Do not justify (ie, leave a 'ragged' right margin).
4. **Do not use type size less than 10 points.**
5. All text, tables, and figures must fit into the rectangle shown.
6. Submit 2 clean copies. This form may be photocopied if multiple abstracts are to be submitted.

Mail original & 1 photocopy (along with postage-paid postcard) to

Respiratory Care Open Forum
11030 Ables Lane
Dallas TX 75229-4593

Early deadline is March 17, 1997 (postmark)

Final deadline is May 27, 1997 (postmark)

Presenter

Name & Credentials

Mailing Address

Voice Phone & Fax

Corresponding Author If Different from Presenter

Name & Credentials

Mailing Address

Voice Phone & Fax

American Association for Respiratory Care Membership Application

Please read the eligibility requirements for each of the classifications in the right-hand column, then complete the applicable section. All information requested below must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

- ☐ Active
- Associate
  - ☐ Foreign
  - ☐ Physician
  - Industrial
- ☐ Special
- ☐ Student

Last Name _____

First Name _____ Middle _____

Social Security No. _____

Home Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Primary Job Responsibility (check one only)

- ☐ Technical Director
- ☐ Assistant Technical Director
- ☐ Pulmonary Function Specialist
- ☐ Instructor/Educator
- ☐ Supervisor
- ☐ Staff Therapist
- ☐ Staff Technician
- ☐ Rehabilitation/Home Care
- ☐ Medical Director
- ☐ Sales
- ☐ Student
- ☐ Other, specify _____

Type of Business

- ☐ Hospital
- ☐ Skilled Nursing Facility
- DME/HME
- ☐ Home Health Agency
- ☐ Educational Institution
- ☐ Manufacturer or supplier
- ☐ Other, specify _____

Date of Birth (optional) _ _____ Sex (optional) _____

U.S. Citizen? ___ Yes ___ No

Have you ever been a member of the AARC? _____

If so, when? From _____ to _____

Preferred mailing address: ☐ Home ☐ Business

FOR ACTIVE MEMBER

An individual is eligible if he/she lives in the U.S. or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994 will continue as such provided his/her membership remains in good standing

Place of Employment _____

_____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Medical Director/Medical Sponsor _____

FOR ASSOCIATE OR SPECIAL MEMBER

Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members. They have all of the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment _____

_____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

FOR STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency.

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member

School/RC Program _____

_____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Length of Program

- ☐ 1 year
- ☐ 2 years
- ☐ 4 years
- ☐ Other, specify_____

Expected Date of Graduation (REQUIRED INFORMATION)

Month_____ Year_____

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • (972) 243-2272 • Fax (972) 484-2720

American Association for Respiratory Care Membership Application

Demographic Questions

We request that you answer these questions in order to help us design services and programs to meet your needs.

Check the Highest Degree Earned

- ☐ High School
- ☐ RC Graduate Technician
- ☐ Associate Degree
- ☐ Bachelor's Degree
- ☐ Master's Degree
- ☐ Doctorate Degree

Number of Years in Respiratory Care

- ☐ 0-2 years
- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 years
- ☐ 16 years or more

Job Status

- ☐ Full Time
- ☐ Part Time

Credentials

- ☐ RRT
- ☐ CRTT
- ☐ Physician
- ☐ CRNA
- ☐ RN
- ☐ LVN/LPN
- ☐ CPFT
- ☐ RPFT
- ☐ Perinatal/Pediatric

Salary

- ☐ Less than $10,000
- ☐ $10,001-$20,000
- ☐ $20,001-$30,000
- ☐ $30,001-$40,000
- ☐ $40,001 or more

PLEASE SIGN

I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics. I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to **Respiratory Care** journal and **AARC Times** magazine includes an allocation of $6.50 from my dues for each of these publications.

NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the nondeductible portion of your dues - the portion which is allocable to lobbying - is 26%.

Signature _____

Date _____

Membership Fees

Payment must accompany your application to the AARC. Fees are for 12 months. (NOTE: Renewal fees are $65.00 Active, Associate-Industrial or Associate-Physician, or Special status; $80.00 for Associate-Foreign status; and $35.00 for Student status.)

☐ Active	$77.50
☐ Associate (Industrial or Physician)	$77.50
☐ Associate (Foreign)	$92.50
☐ Special	$77.50
☐ Student	$35.00
TOTAL	$_____

Specialty Sections

Established to recognize the specialty areas of respiratory care. The sections publish a newsletter four times a year that focuses on issues of specific concern to that specialty. The sections also design the specialty programming at the national AARC meetings.

☐ Adult Acute Care Section	$ 5.00
☐ Education Section	$10.00
☐ Perinatal-Pediatric Section	$ 5.00
☐ Diagnostics Section	$ 5.00
☐ Continuing Care-Rehabilitation Section	$ 5.00
☐ Management Section	$10.00
☐ Transport Section	$ 5.00
☐ Home Care	$ 5.00
☐ Subacute Care	$ 5.00
TOTAL	$_____

GRAND TOTAL = Membership Fee plus optional sections $_____

☐ Total Amount Enclosed $_____

☐ Please charge my dues (see below)

To charge your dues, complete the following:

- ☐ MasterCard
- ☐ Visa

Card Number

__ __ __ __ __ __ __ __ __ __ __ __ __ __ __ __

Card Expires _____/_____

Signature _____

Mail application and appropriate fees to:

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • (972) 243-2272 • Fax (972) 484-2720

MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting by health professionals of adverse events and product problems

Page ____ of ____

Form Approved OMB No. 0910-0291 Expires 4/30/96
See OMB statement on reverse

FDA Use Only (Resp Care)

Triage unit sequence #

A. Patient information

1. Patient identifier	2. Age at time of event: or Date of birth:	3. Sex	4. Weight
In confidence		☐ female ☐ male	____ lbs or ____ kgs

B. Adverse event or product problem

1 ☐ Adverse event and/or ☐ Product problem (e.g., defects/malfunctions)

2. Outcomes attributed to adverse event (check all that apply)

- ☐ death ____ (mo/day/yr)
- ☐ life-threatening
- ☐ hospitalization – initial or prolonged
- ☐ disability
- ☐ congenital anomaly
- ☐ required intervention to prevent permanent impairment/damage
- ☐ other

3. Date of event (mo/day/yr)	4. Date of this report (mo/day/yr)

5. Describe event or problem

6. Relevant tests/laboratory data, including dates

7. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1 Name (give labeled strength & mfr/labeler, if known)

#1

#2

2. Dose, frequency & route used	3. Therapy dates (if unknown, give duration) from to (or best estimate)
#1	#1
#2	#2

4. Diagnosis for use (indication)	5. Event abated after use stopped or dose reduced
#1	#1 ☐ yes ☐ no ☐ doesn't apply
#2	#2 ☐ yes ☐ no ☐ doesn't apply

6. Lot # (if known)	7. Exp. date (if known)	8. Event reappeared after reintroduction
#1	#1	#1 ☐ yes ☐ no ☐ doesn't apply
#2	#2	#2 ☐ yes ☐ no ☐ doesn't apply

9 NDC # (for product problems only) – –

10. Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1 Brand name

2. Type of device

3. Manufacturer name & address

4. Operator of device
- ☐ health professional
- ☐ lay user/patient
- ☐ other

5. Expiration date (mo/day/yr)

6.
model # ____
catalog # ____
serial # ____
lot # ____
other #

7. If implanted, give date (mo/day/yr)

8. If explanted, give date (mo/day/yr)

9 Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ____ (mo/day/yr)

10. Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. Name & address	phone #

2 Health professional?	3. Occupation	4 Also reported to
☐ yes ☐ no		☐ manufacturer ☐ user facility ☐ distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

FDA Form 3500 1/96)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 to report by phone or for more information
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

The public reporting burden for this collection of information has been estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to:

DHHS Reports Clearance Office
Paperwork Reduction Project (0910-0291)
Hubert H. Humphrey Building, Room 531-H
200 Independence Avenue, S.W.
Washington, DC 20201

An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number.

Please do NOT return this form to either of these addresses.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service • Food and Drug Administration

FDA Form 3500-back

Please Use Address Provided Below – Just Fold In Thirds, Tape and Mail

---

Department of Health and Human Services
Public Health Service
Food and Drug Administration
Rockville, MD 20857

Official Business
Penalty for Private Use $300

NO POSTAGE NECESSARY IF MAILED IN THE UNITED STATES OR APO/FPO

BUSINESS REPLY MAIL
FIRST CLASS MAIL PERMIT NO. 946 ROCKVILLE, MD

POSTAGE WILL BE PAID BY FOOD AND DRUG ADMINISTRATION

MEDWATCH
The FDA Medical Products Reporting Program
Food and Drug Administration
5600 Fishers Lane
Rockville, MD 20852-9787

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month in which you wish the ad to run. Submit copy and insertion orders to Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

Calendar of Events

AARC & AFFILIATES

February 2–5—The American College of Chest Physicians (ACCP) and the AARC

Cardiopulmonary Wellness & Rehabilitation meeting in Snowbird, Utah.

Contact: Arlene Karavich, ACCP, 3300 Dundee Rd, Northbrook IL 60062, (847) 498-8358, fax (847) 498-5460.

April 1–4—Indiana, Kentucky, and Ohio Societies

24th Annual Region II for Respiratory Care Meeting, "Winning the Game of Respiratory Care," at the Albert Sabin Convention Center, Cincinnati, Ohio.

Contact: 1-800-691-3046, mailbox #1, or http://www.bright.net/~dsibb/Reg2rc.htm.

April 24–25—Respiratory Care Services of Arkansas Children's Hospital and the Arkansas Society

Diamond Conference in Little Rock, Arkansas. Luncheon tutorials are available.

Contact: Mike Anders at (501) 320-4782, Kathy Boyle at (501) 320-3535, fax (501) 320-3411, or visit http://www.ach.uams.edu/announce/diamond.

April 25—Texas Society's Alamo District, the University of Texas Health Science Center at San Antonio (UTHSCSA), and Wilford Hall Medical Center

Seminar at the San Antonio Municipal Auditorium and Holiday Inn Riverwalk North, San Antonio, Texas.

CRCE: Credit has been requested.

Contact: Respiratory Care, UTHSCSA, at (210) 567-3706.

OTHER MEETINGS

May 16–21—American Lung Association/American Thoracic Society (ALA/ATS)

Annual International Conference, in San Francisco, California.

Contact: 1997 International Conference, ALA/ATS, 1740 Broadway, New York, NY 10019-4374.

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

RESPIRATORY CARE Special Issues

The April 1997 issue of the Journal will feature papers and discussions from the Consensus Conference: Noninvasive Positive Pressure Ventilation held in Vail, Colorado, October 4-6, 1996. The conference was chaired by Neil R MacIntyre MD and Dean R Hess PhD RRT. Authors include David J Pierson MD, Robert M Kacmarek PhD RRT, John R Bach MD, W Gerald Teague MD, Nichols R Hill MD, Gordon D Rubenfeld MD, Richard G Wunderink MD, Dean R Hess PhD RRT, Diane Kreimer RN RRT, and Robert E Turner RRT.

The September 1997 issue of the Journal will be devoted to education—respiratory care formal programs and curriculum, continuing and in-service education, and patient education. Original studies, state-of-the-art reviews, teaching features, reviews of instructional materials, and other areas of education are encouraged. Send your unpublished manuscripts for peer review. For more details, call Kris Williams, Assistant Editor, (972) 406-4665.

Know the Area Code for AARC: 972

Phone: (972) 243-2272
Fax: (972) 484-2720 or (972) 484-6010

Note: If you're having difficulty reaching us at this area code—call your local long-distance provider.

Editorial Office via the Internet

Web browsers can now reach the RESPIRATORY CARE Editorial Office via e-mail. Questions about the status of a manuscript, writing guides, other AARC Web sites, Clinical Practice Guidelines, and more can be answered by our Editorial Assistant, Linda Barcus. Her e-mail address is **barcus@aarc.org**. E-mail addresses for the Editor and the Assistant Editor will be available later in the year.

Helpful Web Sites

American Association for Respiratory Care
http://www.aarc.org

National Board for Respiratory Care
http://www.nbrc.org

Applied Measurement Professionals Inc
http://www.applmeapro.com

Food and Drug Administration
http://www.fda.gov

Center for Devices and Radiological Health
http://www.fda.gov/cdrh/index.html

Tuberculosis Information
http://www.umdnj.edu/ntbc

The Lambda Beta Society
The National Honor Society for the Profession of Respiratory Care

For information about the Lambda Beta Society—
the National Honor Society for the
Profession of Respiratory Care,
contact the Society Office at
1701 W Euless Blvd, Suite 200, Euless TX 76040
(817)283-4269.

The National Board for Respiratory Care—1997 Examination Dates and Fees

Examination	Examination Date	Examination Fee	
CRTT Examination	March 8, 1997 Application Deadline: January 1, 1997	$ 100 60	(new applicant) (reapplicant)
	July 12, 1997 Application Deadline: May 1, 1997	100 60	(new applicant) (reapplicant)
	November 8, 1997 Application Deadline: September 1, 1997	100 60	(new applicant) (reapplicant)
RRT Examination	June 7, 1997 Application Deadline: February 1, 1997	100 60 110	Written only (new applicant) Written only (reapplicant) CSE only (all applicants)
	December 6, 1997 Application Deadline: August 1, 1997	210 170	Both (new applicant) Both (reapplicant)
CPFT Examination	June 7, 1997 Application Deadline: April 1, 1997	110 80	(new applicant) (reapplicant)
RPFT Examination	December 6, 1997 Application Deadline: September 1, 1997	160 130	(new applicant) (reapplicant)
Perinatal/Pediatric Respiratory Care Specialty Examination	March 8, 1997 Application Deadline: November 1, 1996	160 130	(new applicant) (reapplicant)

For information about other services or fees, write to the
National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200, FAX (913) 541-0156, email nbrc-info@nbrc.org.

New Products & Services

News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE Journal, New Products and Services Dept. 11030 Ables Lane, Dallas TX 75229-4593.

CO_2 ANALYZER. CEA Instruments Inc introduces the wall-mounted continuous infrared CO_2 analyzer, ADC 200. According to the company, the analyzer is water and dust resistant and features digital display, adjustable automatic zero cycle, and battery backup. Sample gas is drawn into the unit through a vacuum pump and tested for the standard CO_2 ranges 0-1500 ppm, 0-3000 ppm, 0-2%, and 0-5%. Audible and visual alarms are also included. An optional remote indicator is available. For details about the analyzer, circle Reader Service Number 160.

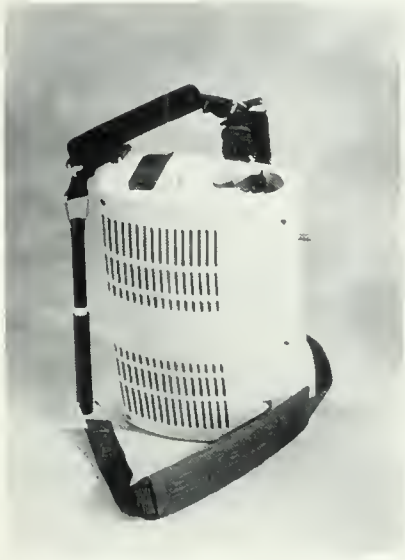
PORTABLE VENTILATOR. Impact Instrumentation Inc reports the addition of the Univent™ Eagle™ Model 754 to its line of products. The Eagle is a small, lightweight (<13 pounds), portable ventilator for use in the critical care setting. The unit contains a compressor, a blender, a graphics display, and alarms, Impact says. Furthermore, the unit features LCD graphs of patient data and device status, apnea mode backup, fixed inspiratory-expiratory ratio, adult, child, or infant operation, manual breath trigger, triggering sensitivity, and disposable ventilator circuits. For a complete list of features or more information, circle Reader Service Number 161.

MOBILE BLOOD ANALYSIS SYSTEM. Diametrics Medical Incorporated now includes the second generation of IRMA® Blood Analysis Systems, the $IRMA_{SL}$. The $IRMA_{SL}$ has an expanded test menu that offers blood gases, electrolytes, and hematocrit testing, according to Diametrics. In addition, the $IRMA_{SL}$ features electronic quality control, enhanced patient information options, and a new technologic platform that provides a base for continued expansion. Options for the $IRMA_{SL}$, such as the IRMA Data Management System version 3.0 and a capillary collection device that makes the system useful in pediatric settings, are available. For information about the system, circle Reader Service Number 162.

QUALITY CONTROL PACKAGE. GAS-ISE™ Survey Validated Blood Gas/Electrolyte QC is offered by Direct Solutions. The GAS-ISE can assess instrumentation bias and the probability of successful proficiency test (PT) performance. Each lot, the company claims, has been validated in one or more PT programs allowing users access to an interlaboratory database for comparison. The package includes a Survey Validation Report that helps identify model-specific groups and CLIA 1988 ranges of acceptability. The package is available in multi-level 108-ampule packs and is a complement to daily controls for instrument validations, correlation studies, troubleshooting, and corrective action procedures. For details, circle Reader Service Number 163.

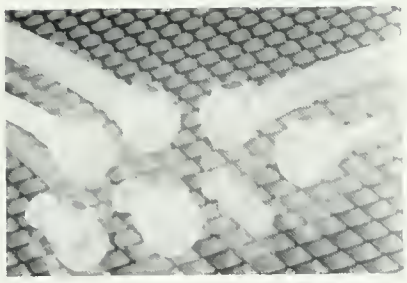
DATA SECURITY DEVICE. E-Systems introduces AuthDisk™, the software authentication device available for both Windows™– and Macintosh®-based remote dial-up clients. The security system offers copy protection and detection and a data encryption standard symmetric challenge-response authentication protocol. The program limits access from modems, data networks, and other uncontrolled media. Only authorized users can access information with a personal identification number. The company lists precise user identification; protection against theft, fraud, and abuse; encrypted software vaulting and electronic fingerprinting; and multi-

platform transparent operation as benefits of the product. For details about the company's service and support package, on-site installation and training, and free upgrades, circle Reader Service Number 164.



PORTABLE LIQUID OXYGEN DELIVERY SYSTEM. DeVilbiss supplies the Pulsair® .5—a compact, portable liquid oxygen delivery system. DeVilbiss says that the Pulsair .5 provides more than 10 hours of oxygen at 2 L/min at 20 breaths/min, weighs about 6 pounds, and features PulseDose® oxygen delivery technology. The Pulsair .5 allows patients to travel longer between refills and is compatible with most liquid oxygen systems. Circle Reader Service Number 165 for more information.

PUBLICATION SUPPLEMENT. Fogg System Company Inc updates the *Guide to Connecting Pressure Transducers* with *Supplement #1*. The new supplement, according to the publisher, lists monitors and transducers that help define the necessary cables for various applications. The company also offers a toll free number for questions. To obtain a free copy of the 16-page *Guide* and *Supplement*, circle Reader Service Number 166.



COUPLINGS. Colder Products Company introduces the MPC Plus Series of quick-disconnect couplings with a locking sleeve for improved fluid line security. According to Colder, the couplings are made of certified USP Class IV material and may be sterilized by electron beam, gamma, ETO, or autoclave methods. The sleeve is available in 1/4- and 3/8-inch clear polycarbonate and white polysulfone. Polycarbonate sealing caps and plugs cover the coupling to prevent fluid line contamination and will not leak with line pressures up to 120 psi. Circle Reader Service Number 167 for information about the MPC Plus Series.

GENERIC INHALATION SOLUTION. DEY Laboratories reports the availability of Albuterol Sulfate Inhalation Solution 0.5%, an alternative to Proventil® and Ventolin®. The solution is AN rated and is indicated for the relief of bronchospasm in patients with reversible obstructive airway disease and acute attacks of bronchospasm. For more information about the nebulizer solution, indications, contraindications, and side effects, circle Reader Service Number 168.

ON-LINE PULMONARY FUNCTION DATA. TeleMedical Inc launches CareConnect ™—a daily pulmonary function test and electronic interview information transmittal system. CareConnect allows the health care provider to collect diagnostic data from lung and heart-lung transplant patients in the clinic or away from the clinic or hospital setting. According to the manufacturer, the CareConnect consists of a Patient Diagnostic Device (a spirometer) that is microprocessor based and capable of performing diagnostic tests, collecting subjective data through an electronic questionnaire, storing the information, and transmitting it via telephone connections to a TeleMedical processing center. The center generates customized Patient Health Reports that are available by fax, modem, mail, or digital media. For specifications, circle Reader Service Number 169.

EXERCISE STRESS TESTING BOOKLET. Vacumetrics Inc publishes an 8-page booklet, *Helpful Hints for Setting Up an Exercise Stress Testing Lab*. The booklet focuses on pulmonary stress testing, reasons for the tests, required equipment, and supplies, the company says. In addition, the booklet offers a table of the performance characteristics of various ventilation measurement devices and lists safety precautions in the exercise laboratory. For a free copy of the booklet, circle Reader Service Number 170.

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Authors in This Issue

Benedetti, Janice....227
Blumer, Jeffrey L....221
Chatburn, Robert L....221
DeAbate, C Andrew....206
Dubbs, William H....204
Durbin Jr, Charles G....218, 227
El-Khatib, Mohamad....221
Faris, Henry....206
Fowler, Cynthia L....206
Graybeal, John M....200
Hagan, Greg....237
Irvin, Charles G....230
Kohl, Edwin C....218
Lasky, Michael S....236
Lawson, Damon R....236
Levine, Edwin R....238
Lough, Marvin....202
Mathewson, Hugh S....234
Mayo, David F....218
McElvaine, Phillip....206
Morgan, Nancy S....206
Nicolls, Mark....230
Pierro, Brian....236
Polak, Elaine M....206
Russell, Melvin....206
Shaw, Robert C....233
Sinkovič, Andreja....214
Uhlemann, Rick....236
Upchurch, John....206
Wanger, Jack....230

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155	156	157	158	159	160	161	162	163	164	165	166	167	168	169
170	171	172	173	174	175	176	177	178	179	180	181	182	183	184
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155	156	157	158	159	160	161	162	163	164	165	166	167	168	169
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Faris, Henry.....
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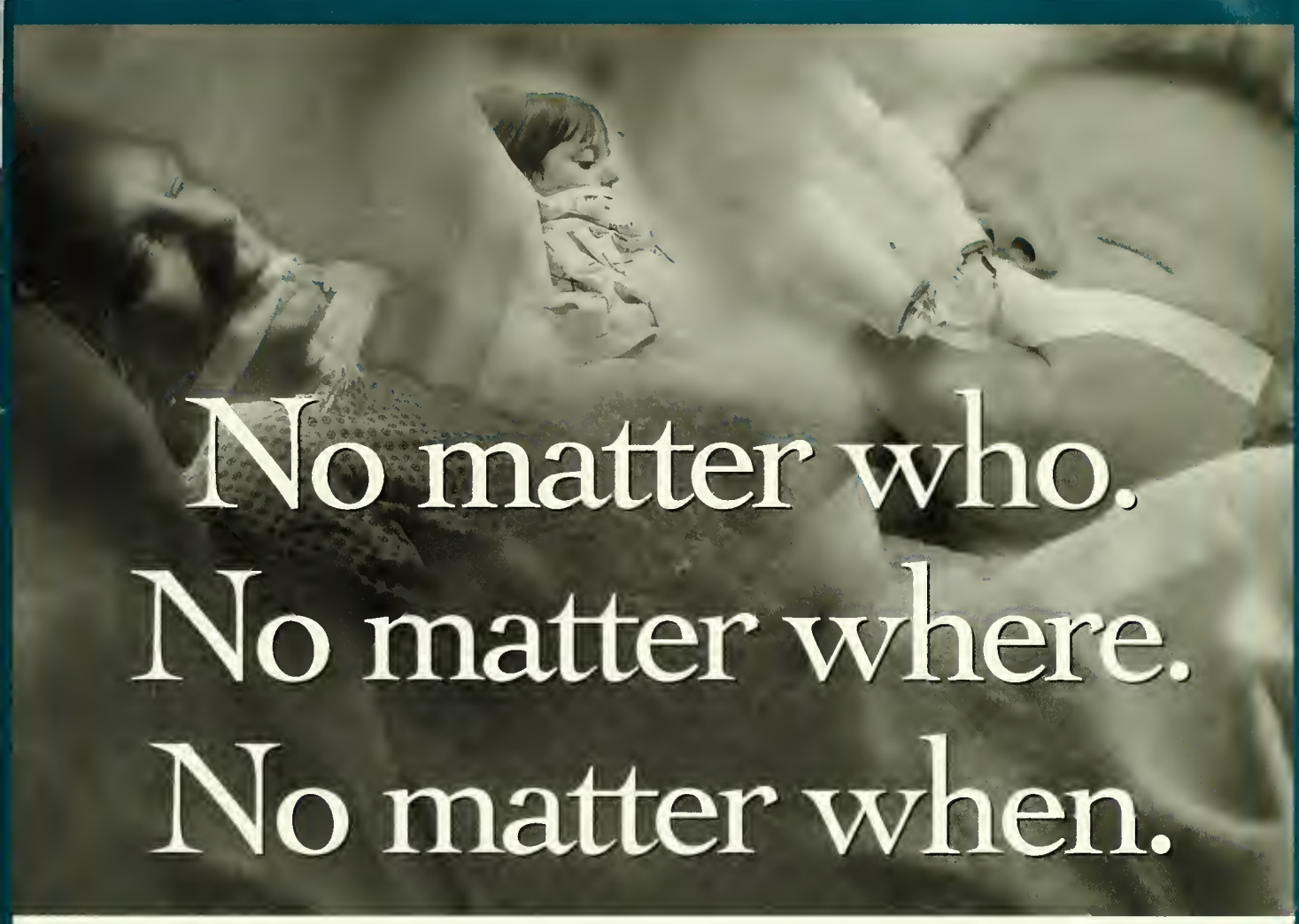

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